Table of Contents

Arterial Disorders	1
Venous Disorders	25
Lymphatic Disorders	39
Peripheral Nerves	47
Thyroid Diseases	54
Neck	82
Breast Diseases	90
Face & Mouth	111
Salivary Disorders	124
Skin & Subcutaneous tissues	130
Hand Infections	142
Muscle & Tendon	147

Arterial Disorders

Arterial Disorders

Peripheral Ischemia

Definition :

Impairment of blood supply to the tissues sufficient to impair their nutrition and function.

Types :

1. Acute ischemia.
2. Chronic ischemia.

The effects of ischemia depend upon :

1. The type of the artery : Some arteries have a very efficient collateral circulation to the extent that their ligature may not be followed by serious sequelae (e.g. the subclavian artery). On the other hand, the brachial and popliteal arteries have poor collateral circulation.

2. The rate of occlusion of the artery : Acute ischemia is much more serious than chronic ischemia as there is no sufficient time for the collateral to develop.

3. The state of the collateral vessels.

4. The general condition of the patient : The presence of myocardial insufficiency or severe anemia will exacerbate the effects of ischemia.

5. Efficiency of treatment.

Acute limb ischemia

Definition :

This serious condition is due to sudden interference with the blood flow to a limb.

Etiology :

1. Embolism : is the commonest cause.

2. Causes in the wall :

(a) Arterial injury.
(b) Acute thrombosis on top of atherosclerosis.
(c) Vasospastic disorders leading to sudden vasoconstriction as in Raynaud's disease.
(d) Intra-arterial thiopentone or cytotoxic substances.
(d) As a complication of aortic dissection.

3. Compression of the artery by : plaster of paris, hematoma, tourniquet, fractured bone.

4. Phlegmasia alba dolens.

Clinical picture :

[1] Type of patient: e.g. history of cardiac disease (embolism) or trauma.

[2] Pain is usually sudden and severe and occurs in 80 % of cases.

- ✓ Pain is absent in some patients because of the rapid onset of anesthesia.

[3] Pallor appears first but is replaced by mottled cyanosis after few hours due to the accumulation of deoxygenated blood.

[4] Paresis passing to paralysis may occur.

- ✓ Paraplegia may be the first presentation of aortic bifurcation embolism.

[5] Parasthesia : Cutaneous hyposthesia progresses slowly to frank anesthesia.

[6] Pulselessness.

[7] Coldness.

Irreversible lower limb ischemia :

[1] Fixed color changes "Blue Staining".

[2] Signs caused by muscle necrosis :

(a) Tense calf.
(b) Fixed plantar flexion of the foot.
(c) Bulging anterior leg compartment.

[3] Acute paraplegia may occur in case of saddle aortic embolism.

Moderate limb ischemia : It occurs if :

1. Embolus does not cause complete occlusion.
2. Pre-existing good collaterals in cases of thrombosis.

- The onset is more gradual and the symptoms are less severe.

Sequelae :

- The sequelae of acute limb ischemia will depend upon the factors mentioned before (Mention them).

Pathological sequelae :

[1] Secondary thrombosis : which extends proximally as far as the nearest branch and distally in the arterial tree including the collaterals.

[2] Muscle compression syndrome : Muscle edema that results from prolonged ischemia obstructs the circulation in the already ischemic limb. Further edema of the muscles that occurs following revascularization obstructs the circulation more and more.

[3] DVT : 2ry to stagnation of blood in veins draining the limb.

[4] Moist gangrene : line of demarcation is several inches below the level of arterial obstruction.

Clinical sequelae :

[1] Complete recovery : if efficient treatment initiated rapidly.
[2] Moist gangrene : line of demarcation is several inches below the level of arterial obstruction.
[3] Picture of chronic ischemia.
[4] Volkmann's ischemic contractures (see orthopedics).
[5] Peripheral edema : Post-revascularization edema can be due to widespread DVT.

Investigations for acute ischemia : Should be urgent :

[A] Imaging studies :

1. **Duplex scan :** localizes and identifies the presence of embolism or a thrombus.
2. **Arteriography :**
   - ✓ It may cause a delay of 2 – 3 hours, therefore, **not done in a threatened limb.**
   - ✓ In **acute embolic ischemia:** it shows sharp cut-off with minimal collaterals.
   - ✓ In **Thrombotic ischemia:** it shows tapering stenosis with extensive collaterals.
   - ✓ Its value is in cases of diagnosed acute thrombosis because it provides information that is essential **before doing an arterial reconstruction.** This information includes :
     - Site of occlusion.
     - Proximal inflow, i.e., if there is another proximal arterial narrowing.
     - Flow distal to the occlusion, i.e. **distal run-off.**
3. **ECG & Echocardiography :** detects cardiac sources of embolism.

[B] Laboratory studies :

- Hemoglobin, urea and Creatinine.
- Acidosis and raised creatine phosphokinase and WBC's indicate extensive muscle necrosis.

Arterial Embolism (Embolic Ischemia & Gangrene)

Definition :

Embolism is a condition in which an embolus is circulating in blood stream & finally impacted in an artery.

- **Embolus is derived from the Greek word that means Plug or stopper.**

Incidence : Usually in a cardiac patient with congenital heart disease, infarction or artificial valve.

Pathology :

[A] Source of arterial embolism : 90 % from the heart

1. From the left atrium as in A.F
2. From the mitral or aortic valve as in S.B.E.
3. From mural thrombus in myocardial infarction.
4. From mural thrombus in an aortic aneurysm.
5. From an atheromatous plaque or thrombus adherent in the aorta or large arteries.
6. Rarely paradoxical embolism may occur in case of A.S.D. or V.S.D.

[B] Site of impaction of an embolus :

- The embolus may be arrested anywhere as the vessel abruptly diminishes in size especially at:
  1. Bifurcation of common femoral artery into superficial and deep femoral arteries (40 %).
  2. The aortic bifurcation **(saddle embolus).**
  3. The bifurcation of the popliteal artery.
  4. The bifurcation of the brachial artery.
  5. The bifurcation of the common carotid artery.

[C] Pathological sequelae : (see before)

- **Usually results in gangrene; Due to :**
  1. **Secondary thrombosis,** which extends proximally as far as the nearest branch and distally in the arterial tree including the collaterals.
  2. **Extensive venous thrombosis,** which aggravates edema and ischemia.
  3. **Reflex spasm** of the vessels and the collaterals.
  4. **Showers of emboli** may block the collaterals.
  5. **The associated heart disease.**

Clinical picture : (see general)

Investigations : E.C.G., Echocardiography and duplex scanning

D.D.:

[1] Embolic from Thrombotic :

Items	Embolic ischemia	Thrombotic ischemia
Age	Commoner in young age	Commoner in elderly patients
Past history of claudications	Absent	Present
Past history of heart disease	Present	Usually absent
Source of an embolus	Present	Absent
Radial pulse	Usually irregular as AF is the commonest cause	Usually regular
Trophic changes	Absent	May be present
Appearance of collateral circulation on angiography	Minimal	Extensive

In addition to differentiation between embolism and thrombosis, other conditions include :

[2] Phlegmasia cerulea dolens.

[3] Aortic dissection.

[4] Low flow states.

[5] Acute occlusion of popliteal and femoral aneurysm.

Treatment :

[I] General measures of treatment :

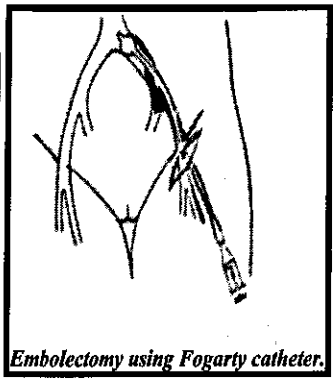
1. Morphine : for pain.
2. I.V. fluids : to correct dehydration, if present.
3. I.V. heparin : Start with 5000 to 10000 IU followed by 3000 – 5000 IU every 2 hours, the dose is controlled by checking activated partial thromboplastin time (APTT) every 12 hours, which should be maintained at 2 - 3 times the baseline level.
4. Care of cardiac condition :

(a) Oxygen if needed.
(b) Lab tests, ECG and chest X-ray.
(c) Digoxin for rapid A.F.
(d) Lasix for heart failure.

[II] Urgent embolectomy :

Under local, spinal or general anesthesia, remove the embolus with the distal and proximal extension thrombus through a transverse arteriotomy using **Fogarty balloon catheter.**

- ✓ Fogarty catheter is introduced until it passes the limit of the thrombus.
- ✓ Balloon is inflated and catheter is withdrawn slowly, together with the clot.
- ✓ The procedure is repeated both proximal and distal until bleeding and back bleeding occurs with palpable distal pulses & operative arteriogram shows patent arteries.
- ✓ Irrigation of the distal arterial tree by heparinized saline.
- ✓ Finally arteriotomy is repaired.



Embolectomy using Fogarty catheter.

Prognosis :

Embolectomy should be carried as soon as possible (within 6 hours) to avoid muscle necrosis and secondary thrombus formation.

- As long as these muscles are soft and the capillary circulation is preserved before the operation, the success rate is over 95% regardless the duration of ischemia.
- If calf muscles necrosis occurs the muscles are rigid, with loss of capillary circulation, the value of embolectomy **(Delayed Embolectomy)** is that when gangrene occurs, the level of amputation will be lower.

N.B.:
1. A completion angiogram should ideally be done at the end of embolectomy to be sure that all emboli have been removed.
2. Heparin should be continued postoperatively until the **cardiac condition** is assessed and the need for further heparin therapy is determined.
3. The source of arterial emboli should be corrected if possible.
4. For aortic bifurcation embolism embolectomy is done via a bilateral femoral arteriotomies.

Complications of embolectomy :

1. Sudden death may occur due to pulmonary embolism secondary to detachment of a large thrombus in the femoral vein. It is therefore advisable to do either:
(a) Thrombectomy of common femoral vein if a thrombus is felt in the femoral vein at operation.
(b) Ligation of the common femoral vein.
(c) Putting a vena-caval filter.
2. Compartments compression. Fasciotomy is done to relieve the tension.
3. Reperfusion injury of the kidney and heart may occur due to the passage of large amounts of myoglobin and potassium from the ischemic muscles.
4.complications of Fogerty's catheter: dissection, vessel rupture, endothelial damage and distal embolization.

[III] Fibrinolytic agents :

Streptokinase, urokinase or tissue plasminogen activator are tried to dissolve embolus without surgery.

- ✓ Streptokinase is an inexpensive but may cause anaphylaxis.
- ✓ Urokinase is an expensive agent, which is a direct plasminogen activator.
- ✓ Tissue plasminogen activator is neither pyrogenic nor antigenic but very expensive.

Technique of direct thrombolysis :

- At the completion of arteriography the tip of the arterial catheter is embedded in the thrombus.
- The usual dose is 5000 unit/hour.
- Monitoring every 6 hours by APTT and fibrinogen titer.

Complications of direct thrombolysis :

- Anaphylactic reaction with the use of streptokinase.
- Hemorrhage at the puncture site, GIT or cerebral.

[IV] Amputation :

Indication : Established gangrene.

N.B the blue toe syndrome:
Atherosclerotic aorta commonly aorto-iliac disease with embolization to toes which will be ischemic and blue while pulses are intact

Acute arterial thrombosis

Arterial thrombosis can occur in the following conditions :

1. On top of chronic occlusive arterial disease, e.g. atherosclerosis (the commonest) or Burger's disease.
2. As a complication of arterial aneurysm.
3. As a result of massive DVT.
4. As a result of traumatic contusion of vessels.
5. May complicate febrile illness or gastroenteritis especially in children. Dehydration leads to hemoconcentration with consequent thrombosis.

Treatment : ***Severe ischaemia.***:Urgent arteriography is performed to plan for emergency revascularization surgery.

- ***Moderate ischaemia.*** Urgent arteriography is performed. There is time for the use of thrombolytic therapy. After dissolution of the thrombus elective revascularization surgery is done. Elective surgery carries a better prognosis that emergency surgery in these cases.

Arterial Injuries

Incidence :

Arterial injuries are increasingly common nowadays due to :

1. The increased motor car accidents and war injuries.
2. Iatrogenic injuries are now increasing with the introduction of invasive techniques, for the diagnosis and management of cardiovascular disorders.

Etiology :

1. **Penetrating injury** e.g. stabs, bullets passing through or near-by an artery **"near miss".**
2. **Blunt injuries** as in road traffic accidents.
3. **Plaster or tourniquet compression.**
4. **Fracture or dislocation.**
5. **Iatrogenic :** Following arterial cannulation.

Pathological types :

[A] Arterial injury with division :

1. Complete division : *Transection injury presents with ischemia rather than hemorrhage*

- Both ends bleed profusely but soon bleeding tends to stop spontaneously & the divided stumps retract
- There is loss of distal pulse & manifestations of ischemia appear.

2. Partial division :

- The contraction and retraction of the vessel wall cause the tear in the vessel wall to gap.
- The artery will continue to bleed either externally or internally producing a pulsating hematoma.
- Blood flow although diminished is not interrupted and keeps the viability of the part.
- The distal pulse is palpable.

[B] Arterial contusion with or without superadded thrombosis :

- The outer coats remain intact.
  If the trauma is not severe, damage occurs within the arterial wall with an intact intima.
- Unless contusion is associated with thrombosis, it cannot be diagnosed except during operation.

[C] Arterial spasm :

- There is no much change in the vessel wall from outside.
- Intimal tear may be present. Spasm without any pathology is very rare and should not be diagnosed as a cause of loss of distal pulse and ischemia except after exploration.

[D] Intra-arterial drug injection. (see later)

Clinical features and assessment of arterial injuries :

[A] History of trauma.

[B] General examination : to look for other injuries & vital signs to detect amount of blood loss.

[C] Local examination : is the basis for diagnosing arterial injury.

I- Hard signs : These are sure signs of arterial injury.

1. External arterial bleeding.
2. Loss of distal pulses.
3. Any of the classic manifestations of acute ischemia.
4. Pulsating or expanding hematoma.
5. Palpable thrill or audible bruit heard with a stethoscope at or distal to the area of injury.

II- Soft signs : These are less specific (equivocal) signs.

1. Small or moderate sized hematoma that is not pulsating and not expanding.
2. Proximity of penetrating wound to a major vascular structure.
3. Adjacent nerve injury, producing neurological deficit.
4. Presentation with shock that can't be explained by other injuries. In these cases the patient could have bled from an arterial injury & the bleeding could have stopped before hospital admission.

Investigations :

In patients with hard signs :

➧ Immediate surgical exploration is indicated without any diagnostic studies.

In patients with soft signs : Urgent investigations are needed :

1. Plain X-ray : to detect foreign bodies or fractures.
2. Arteriography : is the most accurate diagnostic study in these cases. A traumatic arterio-venous fistula is detected by early venous filling.
3. Duplex ultrasonography : can be a reliable substitute.
4. If the soft sign is "shock" no diagnostic studies should be done. Resuscitation is the first priority. Arteriography can be done in the operating room.

Treatment :

[A] Immediate treatment for arterial injuries :

1. Control of external bleeding. (See shock).
2. **Resuscitation** by blood and IV fluids.
3. **Heparinization** : Systemic heparin is recommended *only* in patients with isolated vascular injury presenting with ischemia.
4. Prophylactic **antibiotics**.

[B] Definitive treatment :

- Exploration is mandatory if the pulse is not felt after reduction of fractures.
- The chances of success of surgical treatment diminish after six hours of ischemia.
- After exposure of the artery, if it is divided or contused, proximal & distal control is applied.
- Injured main veins are repaired before the arteries.
- Local heparin should be used routinely.
- Development of swelling after revascularization is an indication for fasciotomy.

1. In clean-cut arterial injuries :

(a) If there is a longitudinal incision or a transverse incision less than one half of the circumference, the injury is repaired.

(b) If more than half of the circumference is injured, complete division is performed & then direct anastomosis is done.

2. If the artery is contused or divided :

- Damaged edges or segments are excised.
- The proximal and distal segments are cleaned from clots by **Fogarty catheter,** flushed with heparinized saline, and then clamped again.
- The injured area should be repaired by **Patch Angioplasty** (an autogenous vein graft or less often a synthetic graft).

3. If the artery is found spastic :

- Local application of papaverine and intra-arterial injection of heparin in saline.
- If not successful, forcible dilatation using Fogarty catheter is performed.
- If spasm still persists, excision of spastic segment with a saphenous vein graft is performed.

- ✓ Damaged main **veins** should be repaired.
- ✓ Damaged **nerves** can be repaired or left for another setting.
- ✓ Distal **fasciotomy** is usually added.

Intra-arterial drug injection

- Due to wrongly injected drugs into arteries instead of veins. The most commonly punctured arteries are the brachial and the radial arteries. The block occurs in the small distal arteries of the digits.

Clinical features :

- Burning discomfort extending from the point of injection to the tips of the fingers immediately after injection, rapidly followed by severe pain and blanching.
- Edema develops rapidly and may be severe.
- Coldness and cyanosis soon follow.
- The distal pulses are often normal.
- Digital gangrene and less commonly total extremity gangrene may then develop.
- Arteriography shouldn't be done as it won't add any more information to the clinical assessment.

Treatment : Should start immediately :

1. Heparin intravenously 10000 I.U followed by continuous infusion
2. Dexamethazone 4mg IV every 6 hours.
3. Low molecular weight dextran (40) minimizes platelet aggregation.
4. Strong analgesics.
5. Extremity elevation to reduce edema.
6. Early passive and active exercises.

In cases of intra-arterial thiopentone injection, anesthetist should not remove the needle from the artery before injecting the heparin bolus intra-arterially

Chronic Ischemia

Definition :

Slowly progressive arterial obstruction.

Etiology :

[A] Above the age of 45 years : atherosclerosis is the commonest cause.

[B] Below the age of 45 years :

1. In diabetics :

- Presenile atherosclerosis is the main cause.

2. In non diabetics :

- In females : Raynaud's disease in the upper limb & arteritis in both upper & lower limbs.
- In males : Burger's disease in the lower limbs and arteritis in both upper and lower limbs.

[C] Other causes :

1. Vasospastic disorders : Ergotism, acrocyanosis, and erythrocyanosis frigida.
2. Aneurysm.
3. Incomplete recovery after treatment of acute ischemia.
4. Vascular compression :
   (a) Thoracic outlet syndrome (in the U.L.).
   (b) Compression by tumor.

Pathology of atherosclerosis :

1. **Atheroma** is the primary pathology of this disease, which starts as an elevated yellow plaque on **the intimal surface** of the arteries.
2. **The subintimal plane** shows accumulation of connective tissue matrix and lipids.
3. **The media** shows fibrosis of the smooth muscles and degeneration of the elastic tissue
4. **The adventitia** shows fibrosis.

Site :

Atheromas most commonly occur adjacent to arterial bifurcations, at the origin of major arterial branches and at sites where an artery passes beneath or through a fascial sling.

Risk factors :

1. Hypertension, hypercholesterolemia, cigarette smoking.
2. Diabetes mellitus, hypertriglyceridemia, obesity.
3. Sedentary or stressful lifestyle and a positive family history.

Distribution :

In descending order of frequency they affect :

1. The coronaries.
2. The cerebrals.
3. Arteries of the lower limbs.
4. The renals.
5. Superior mesenteric artery.
6. Celiac artery.
7. Arteries of the upper limbs.

Complications of atherosclerosis:

1- Chronic ischaemia due to gradual arterial occlusion.
2- Acute ischaemia.
3- Aneurysm formation.

Clinical picture :

General examination of a case of chromic ischemia

1. All accessible arteries are palpated to assess the **pulse** and the condition of the arterial wall.
2. **Blood pressure** should be measured.
3. Stethoscope over stenosed artery may reveal a

[1] Pain :

The main presentation

(A) Intermittent claudication :

- **The earliest symptom: Cramping pain, occurs on walking & relieved by rest**
- **The claudication distance :** the distance which the patient can walk until pain occur.
- **The claudication time :** the time which the patient can walk until pain occur.
- **As the disease progress,** the claudication distance gradually shortens and the period of rest increases in length.
- **Site of pain :** depends on site of obstruction (one joint distal to the site of obstruction).
- **Mechanism :** Accumulation of metabolites due to muscle ischemia during walking. These metabolites are gradually washed away during rest.

(B) Rest pain :

- **A continuous burning pain** occurs during rest, which is maximum at night in the forefoot and toes.
- **It is worsened by elevation of the limb.**
- At first relieved by hanging the limb by the side of the bed and exposing it but in severe cases analgesics cannot relieve it completely.
- It occurs in advanced cases due to **ischemic neuritis (nerve ischemia).**

[2] Trophic changes :

1. Skin : Atrophic, thin, dry, scaly, inelastic with loss of hair. Ischemic ulcers begin over the toes or foot.
2. Loss of subcutaneous fat & thin tapering toes.
3. Nails : Loss of luster, fissured, brittle, deformed.

[3] Skin temperature : The level of coldness indicates the level of ischemia.

[4] Color changes :

(A) Mild ischemia : color may be normal.
(B) Moderate ischemia : postural color changes, pallor on elevation **(Buerger's angle)** and **Rubor** on dependency.
(C) Severe ischemia : Fixed color changes : Pallor, cyanosis & redness **(Rubor).**

- The cyanosis & rubor are due to stagnation of blood in the markedly dilated capillaries under the effect of accumulated vasodilator metabolites. The color of blood is at first red (Rubor) later on it becomes blue due to extraction of oxygen by tissues.

[5] Arterial pulsations :

(a) Weak pulsation (partial obstruction or in presence of good collaterals). **OR :**
(b) Absent pulsation (complete obstruction).

[6] Motor and sensory changes : Muscle weakness, hyperesthesia or sensory loss.

[7] Gangrene in severe ischemia.

[8] Impotence : may occur in aortic bifurcation block **(Le Riche Syndrome)** due to diminished blood flow in the internal iliac arteries.

N.B:
Leriche syndrome is a triad of claudication in one or both lower limbs, reduced sexual potency in male patients and diminished or absent femoral pulses.

- **Claudication : From the word : Claudications = to limp.**
  (The Roman emperor Claudis, Walked with a limp due to polio).
- **Pseudo claudication : It is due to neurospinal compression.**

Symptoms increases by lumbar lordosis, so standing or walking causes symptoms which are not relieved till the patient sits and straightens his Lumbar spine.

- **Rest pain :**

1. Relieved by standing erect as gravity aids the delivery of arterial blood to L.L.
2. Classically, it is preceded with claudications but may occur de-novo in patients whose walking is limited by other diseases (e.g. Angina pt).

- **Although ischemia affects both L.L., the patient feels pain in one limb only (the more affected) as he stops walking before developing pain in the less affected limb. Similarly a patient with generalized atherosclerosis may present only by angina pain, which forces him to stop walking before developing claudication in his limbs.**

Special tests :

1. Capillary circulation test :

Note the effect of pressure on the nail bed or skin.

- **Normally** pressure causes blanching & release of pressure is followed by immediate return of normal color.
- **In ischemia,** slow return of normal color indicates sluggish capillary circulation and failure of blanching indicates that the part is gangrenous.

2. Harvey's venous refilling test :

- **Normal :** venous filling occurs in 10-15 seconds.
- **In mild ischemia :** delayed up to 30 seconds.
- **In severe ischemia :** delayed more than 2 minutes.

Clinical Staging of chronic limb ischemia :

Stage I	Asymptomatic.
Stage II	Intermittent claudication.
Stage III	Rest pain.
Stage IV	Ulceration or gangrene.

Differential diagnosis :

I- Atherosclerosis & Buerger's disease :

Items	Atherosclerosis	Buerger's disease
Age of start	Elderly	20 – 40 years
Sex	Usually in males, possibly females	Only males, never females
Etiology	Main risk factors are hypertension, high cholesterol & DM	Excessive smoking
Level of lesion	Aorto-iliac, femoro-popliteal or distal	Distal vessels with patchy distribution
Pathology	Mainly intimal (atheroma)	Inflamed neurovascular bundle & thrombi that block lumen
Rest pain	Late	Early
Migrating thrombophlebitis	Absent	Usually present

II- Other causes of chronic pain in lower limb.

Investigations :

I- Laboratory investigations :

[A] Urine : For sugar.

[B] Blood :

1. Full blood picture for anemia, polycythemia (increased blood viscosity).
2. Blood sugar curve for D.M.
3. Blood urea and renal function tests.
4. Serum cholesterol and lipid (high in atherosclerosis).

II- ECG and fundus examination.

III- Radiological investigations :

[A] Doppler Ultrasound : Values :

1. It is possible to map out obstructions or stenosis over arteries.
2. The systolic pressure can be measured even where the arterial pulse is not palpable.
3. Helpful in observing the success of arterial reconstruction.

N.B: ***normal arterial flow gives a triphasic wave and the collateral flow gives a biphasic wave.***

[B] Measurement of Ankle-brachial pressure index (PI) : (by using the doppler)
The ratio of pressure measured in the posterior tibial artery at the ankle, to the pressure in brachial artery. **The resting PI is greater than 1.**

- If the ratio is < 0.9 it denotes ischemia.
- If < 0.7 = severe ischemia
- If < 0.3 = impending gangrene.

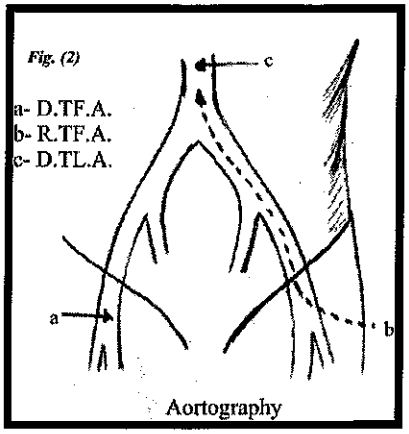
[C] Duplex scan : it visualizes the arteries & it can detect stenosis.

[D] Arteriography : Main method in diagnosis.

Indication :
It is done only after the decision of surgery has been under taken.

Values : Shows :
1. The condition of the arteries.
2. Site and extent of any obstruction.
3. Efficiency of collateral circulation.
4. Peripheral run off (distal blood flow beyond the occlusion). This is the most important as it determines the possibility of direct arterial surgery.

Methods :
1. Direct percutaneous femoral arteriography.
2. Aortography :
(a) Retrograde transfermoral aortography using **Seldinger's needle.**
(b) Direct translumbar aortography.
(c) Transbrachial aortography : If the whole distal aorta is occluded.



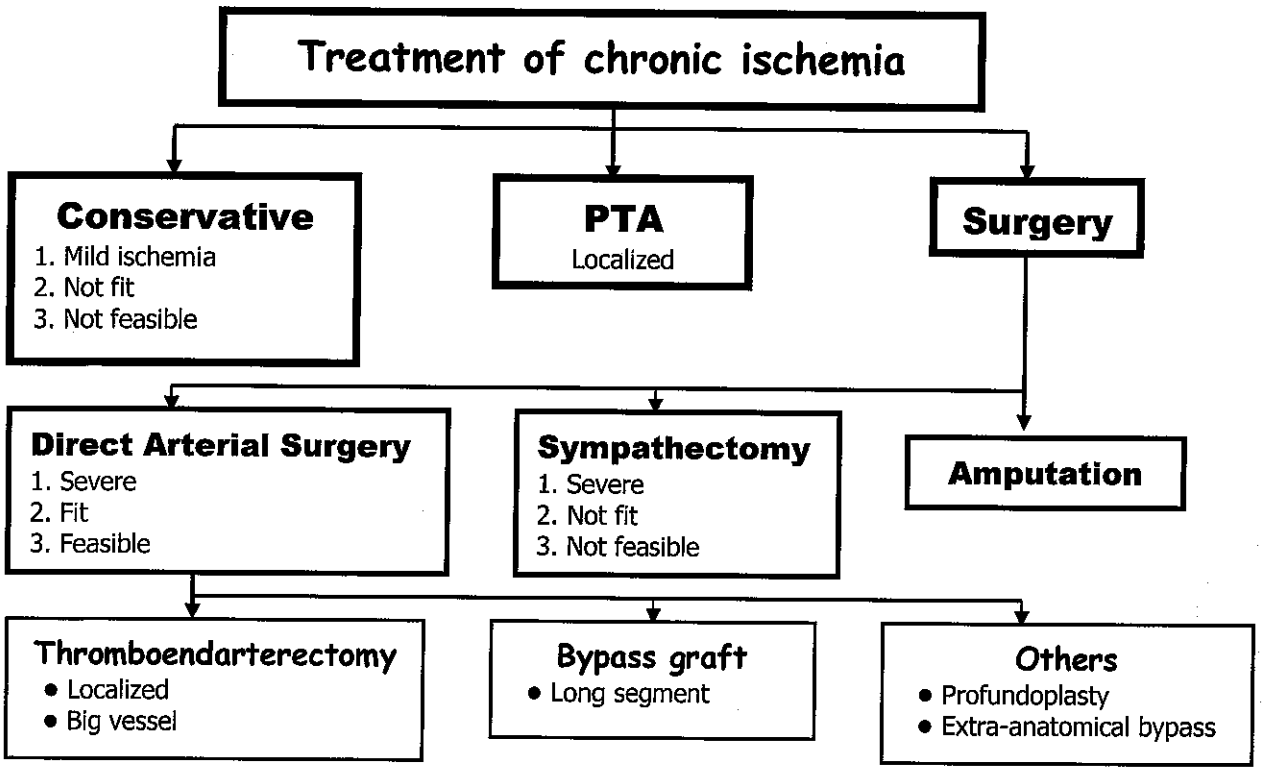
Hazards :
1. Anaphylaxis.
2. Hemorrhage.
3. Spasm.
4. Thrombosis.
5. Dislodgement of atheromatous plaque + embolization.
6. Dissecting aneurysm.

[E] Digital subtraction angiography (DSA): Less invasive than the conventional arteriography. An X-ray picture is taken and the image is introduced to the computer. An intravenous injection of contrast material is given. The contrast will travel to the heart and then to the arteries. Further X-rays are taken

and the images are computerized. By subtracting the first image from the second an arteriogram can be obtained.

[F] Computed tomographic angiography (CTA): allows accurate 3D imaging of arteries or aneurysm. It gives pictures similar to those obtained by conventional angiography. Yet without the need for cannulation of the vessels.

[G] Magnetic resonance angiography: this technique can visualize blood vessels without the injection of any contrast and it can provide a three dimensional image of the examined vessels.



I- Conservative Methods :

Indications :

1. Mild ischemia.
2. If the general condition of the patient is poor.
3. When an operation is not technically feasible.

It includes :

1. Mild exercise short of causing pain (help the collateral circulation).
2. Smoking must be stopped.
3. Correction of anemia.
4. Correction of DM, hypertension and hyperlipidemia.
5. Drugs, which prevent platelet aggregation as dipyridamole (persantin), small dose of aspirin, pentoxiphylline or PGE1.
6. **Protection of ischemic parts :**
   (a) Carefully washed, dried, and powdered.
   (b) Protected by woolen socks (in winter) and suitable shoes.
   (c) Nails and corns are cut cautiously.
   (d) Infections (bacterial or fungal) are treated properly.

II- Interventional radiological procedures :

Indications :

Localized obstruction in large and medium sized arteries.

Methods :

1. Percutaneous transluminal angioplasty (PTA) : a special balloon catheter is introduced percutaneously until the balloon reaches the stenosed or occluded segment. It is then inflated to dilate the stenosed segment.

2. Application of stent : After balloon angioplasty, a stent can be inserted.

3. Destruction of the atheroma by laser : can be performed before angioplasty.

III- Surgical treatment :

Indication of surgery in a case of chromic ischemia

1. **Sever claudications that interfere with the patient's work.**
2. **Critical ischemia (ischemic ulcer, rest pain or minor gangrene)**

[A] Direct arterial surgery : (Arterial reconstruction)

It should be noted that these operations are **salvage procedures** & should not be performed for early mild cases (e.g. intermittent claudication) as if the operations fails loss of the limb may occur.

Indications :

1. Severe ischemia (rest pain or pre-gangrene).
2. Adequate run off.
3. Proximal arterial occlusion (anywhere from abdominal aorta to the terminal part of popliteal artery).
4. Good general condition.

N.B.: Claudication is a relative indication for surgery e.g. if the patient may lose his job, if he cannot walk for a long distance.

Method :

1. Thromboendarterectomy :

Indication :

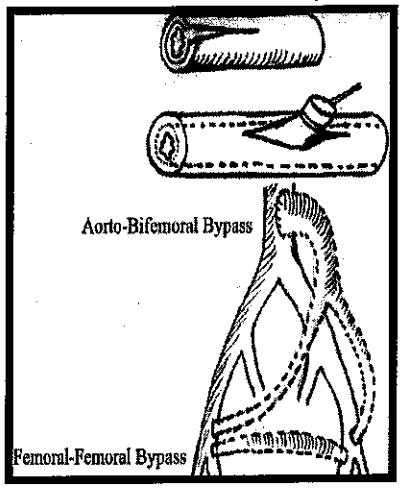
Large artery (e.g. aorta and common iliac) and localized lesions.

- Removal of the thickened atherosclerotic intima with the overlying thrombus using endarterectomy loop. Before closing the arteriotomy incision, the distal intima should be attached to arterial wall by interrupted sutures to prevent its dissection later on.

2. Bypass grafting :

Indication :

Big and medium sized artery with extensive or multiple lesions involving a long segment.



Types of arterial grafts :

(a) Synthetic grafts : Teflon, Dacron or Polytetrafluroethylene (PTFE).
Indication : Large arteries e.g. Aorto-iliac segment.

(b) Autogenous grafts : Using long Saphenous. It is either :

(i) Reversed long Saphenous vein graft : to prevent obstruction by its valves. It is the graft of choice for femoro-popliteal segment.

(ii) In situ long saphenous vein graft : valves are destroyed using special valve strippers. All tributaries are ligated to prevent A.V. fistula.

[B] Indirect arterial surgery : (Lumbar sympathectomy)

Indications :

1. Cases of ischemia when direct arterial surgery is **NOT** feasible i.e. with distal occlusion (no good distal run-off).

It is of value in cases :

(a) With ischemic ulcers. **And**
(b) With rest pain.

2. Many surgeons combine lumbar sympathectomy with direct arterial surgery.
3. After amputation to help wound healing.

Contraindications :

1. Intermittent claudication (worsen the muscle ischemia).
2. Gangrene (ineffective).

[C] Amputation :

There are 2 possibilities :

1. Conservative Amputation :

- If blood supply of adjacent tissues is good or can be improved, line of demarcation appears and separation proceeds by aseptic ulceration.

- **In such cases do either :**

1. Excision of toes at line of demarcation leaving raw surface to heal by granulation.
2. Transmetatarsal or midtarsal amputation in forefoot gangrene provided there is viable long plantar flap reaching base of toes, to cover the stump (dorsal skin cannot withstand trauma).

2. Urgent high amputation :

Indicated for :

1. Spreading gangrene endangering the patient's life.
2. Uncontrollable infection and toxemia.
3. Severe pain deteriorating the general condition of the patient.

Level of amputation :

Depends on the blood supply sufficient for healing of the wound :

- In atherosclerosis (involve femoral artery) above knee amputation (the stump will be supplied by the profunda femoris).
- If popliteal pulse is felt below knee amputation.

Arterial Aneurysm

Definition :

Pathological definition A sac that contains blood and communicates with the lumen of an artery.

Practical definition a permanent localized dilatation of an artery, having at least **1.5** times the normal diameter of that given segment.

They can be classified according to: *I, II and III.*

I- Etiology :

[A] Pathological : The commonest causes :

1. Atherosclerosis : (the commonest cause nowadays).
2. Syphilis affecting aortic arch.
3. Subacute bacterial endocarditis → infected emboli → mycotic aneurysms.
4. Collagen diseases e.g. Behcet disease, Marfan's syndrome and Ehler's Danlos syndrome.

[B] Traumatic :

1. Weakening of the wall of the artery and dilatation later **(true aneurysm).**
2. Interruption of the wall leading to pulsating hematoma which when organized forms an aneurysm **(false aneurysm).**

[C] Congenital : Commonest in circle of Willis → subarachnoid hemorrhage.
The splenic, renal and celiac vessels are possible sites of arterial aneurysms

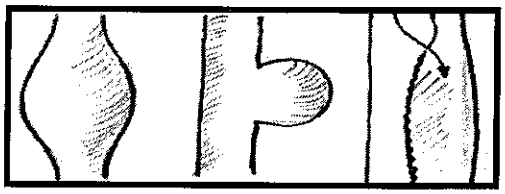
II- Structure of the aneurysm :

1. True aneurysm : The wall of the aneurysm is formed by the stretched wall of the artery (3 layers).

2. False aneurysm: The wall is formed of fibrous tissue due to organization of pulsating hematoma.

III- Shape :

1. Fusiform 2. Saccular 3. Dissecting aneurysm



Clinical picture :

[A] May be silent.

[B] General examination : Manifestations of the cause e.g. syphilis, atherosclerosis...etc.

[C] Swelling which is :

1. Along the course of an artery
2. ***Expansile pulsations*** synchronous with the heart beats *The most important sign*
3. Moves side to side across but not along the course of the artery
4. Compressible (not compressible if filled with a thrombus), smooth, rounded
5. Proximal pressure reduces the size and pulsations
6. Distal pressure increases the size of the aneurysm
7. There is a systolic thrill on palpation, which propagates distally
8. Auscultation: Systolic bruit

[D] Compression on adjacent structures :

Veins Edema due to compression of main veins.
Nerve Compression of a nerve leads to pain, sensory loss and paralysis.
Bone Adjacent bone may be eroded.

[E] Picture of complications :

1. **Rupture :** The most serious
2. **Thrombosis.**
3. **Embolization** due to detached thrombi from the aneurysm leading to distal ischemia.
4. **Pressure on the surrounding structures** (artery, veins, bones, nerves...etc).
5. **Infection :** May lead to secondary hemorrhage.
6. **Distal ischemia due to :**

(a) Thrombosis of the aneurysm. (b) Compression of the main artery.
(c) Embolization. (d) Associated atherosclerosis.

Differential diagnosis :

1. Transmitted pulsation :

- Not expansile & pulsation disappear if you push the mass away from the course of the artery.
- No decrease in size of the swelling with proximal compression.

2. Pulsating tumors e.g. vascular sarcoma :

- Irregular in shape and consistency, not compressible, ill defined.
- Do not overlie the line of a main artery.

3. Arteriovenous fistula :

- Pulsating V.V.
- Machinery murmur and thrill, which propagates distally and proximally.
- Tachycardia and Branham's sign.

4. Acute abscess.

5. A serpentine artery : is sometimes the case as with the common carotid.

Investigations :

1. Plain X-ray :

(a) Eating up of nearby bone.
(b) Calcification in cases of atherosclerosis.

2. Duplex scanning.

3. Abdominal ultrasound :

(a) It is the investigation of choice in aortic aneurysm.
(b) It gives an accurate idea about its size.

4. CT scan :

a) It can demonstrate the relation of the aneurysm to the renal vessels.
b) It can visualize leaking aneurysms.
c) Spiral C.T is superior to the conventional axial C.T.

5. Magnetic resonance arteriography :

- It is a good alternative to CT-scan but is more costly in diagnosis of abdominal aortic aneurysm.

6. Conventional angiography and digital subtraction angiography (DSA) :

- Not routinely needed.

Angiography is neither used to diagnose the aneurysm nor to assess its dimensions

- **Value** :

(a) Visualize the relation of the aneurysm to the renal or mesenteric vessels.
(b) Visualize the distal arteries.

N.B.: An angiogram might appear normal if the aneurysm is occupied by a thrombus.

Treatment :

- Aneurysms are very liable to rupture.
  So, any aneurysm larger than 4 cm (5 cm in case of aortic aneurysm) should be treated surgically.

1. The standard line of treatment is excision and graft :

- The aneurysmal sac is excised.
- The continuity of artery is restored by arterial grafting either by autogenous vein graft or synthetic graft made of Dacron.

However other surgical techniques may be performed:

2. Insertion of a graft inside the sac of the aneurysm without removal of the sac :

- This is commonly done in aortic aneurysm
  (The graft is anastomosed to the aorta above and below the sac)

3. Excision and ligation of proximal and distal arteries :

- Can be performed in aneurysms of radial or ulnar arteries and other small arteries.

Abdominal Aortic Aneurysm

Incidence : This is the commonest type of arterial aneurysms.

Site : Below the origin of the renal arteries in 95 % of cases. It may extend to affect the iliacs.

Etiology : Atherosclerosis is the cause in 95 % of cases.

Clinical picture : As general.

- ✓ *75% of cases are accidently discovered.*
- ✓ *Pain is the commonest symptom (in back & flanks)*
- ✓ *Rupture is the most frequent complication*
- ✓ *The classical triad of rupture includes:*

sudden sever pain, pulasating abdominal mass & shock

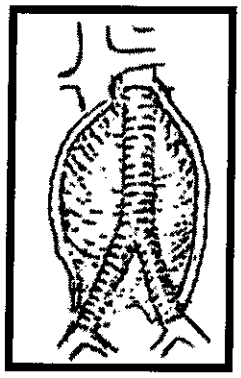
Complications :

As usual + Rupture or leakage

a) **Intraperitoneal rupture (20 %)** is rapidly fatal
b) **retroperitoneal rupture (80 %)** fortunately the patient can be saved

The patient presents by : hypovolemic shock, severe abdominal pain and backache and rapidly enlarging, pulsating mass in the lumbar region.

Investigations : As general.



Treatment : As general.

Plan of management :

1. **Immediate surgery** for patients with the diagnosis of rupture.
2. **Urgent surgery** for patients with symptoms of acute expansion.
3. **Elective surgery** for :
   (a) Symptomatic aneurysms regardless of the size.
   (b) Asymptomatic aneurysms 5cm or more in diameter.

Type of surgery :

1. The standard is conventional open surgery :

- By opening the aneurysm and excluding it by implanting a synthetic graft.

2. Endovascular repair :

- This technique is expensive and recurrence of the aneurysm may occur and indicated for :
  (a) High risk patients.
  (b) High-risk local abdominal factors.

Aortic dissection

Predisposing factors : Atherosclerosis and hypertension.

Pathology :

- An intimal tear allows blood to dissect its way in the media in a caudal direction.
- Blood in the false lumen may rupture inside the true lumen or may rupture to the outside causing fatal hemorrhage. As blood dissects its way distally it occludes the ostia of aortic branches causing ischemia.

There are two main types :

- **Type A :** more serious type. Dissection starts in the ascending aorta. It is likely to produce aortic incompetence, coronary artery occlusion and fatal cardiac tamponade.
- **Type B :** Dissection starts distal to the arch.

Clinical features :

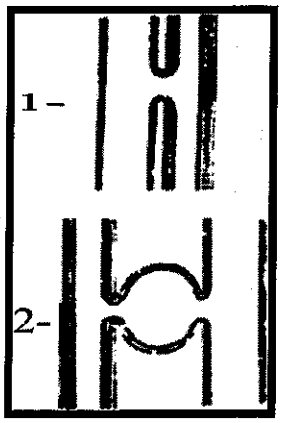
- Severe chest pain radiating to interscapular region similar to that of myocardial infarction.

Investigations : ECG, CT chest, aortography & Echocardiography.

Treatment :

- **Type A** requires urgent surgery by a synthetic graft at the aortic root.
- **Type B** is treated by antihypertensives. Elective surgery may be needed in cases of chronic extension of dissection.

A-V Fistula



Definition : Abnormal connection between an artery and a vein.

Pathological types :

1. Aneurysmal varix : Direct communication between an artery and a vein.
2. Varicose aneurysm : The artery and vein communicate through a false sac.

Types :

[A] Congenital : Usually small, multiple, deep in the limbs leading to :

- Local gigantism, edema, congestion, pulsating V.V. and venous ulceration.
- Multiple haemoangiomata are common.

[B] Acquired : Due to penetrating trauma.

- **Local signs : Swelling :**

1. Cystic, smooth, rounded & compressible.
2. Along the course of an artery.
3. Proximal compressing reduce its size and pulsation.
4. Continuous thrill & machinary murmur which propagates both distal and proximal.
5. No local gigantism.

- **Distal signs :** Congestion, edema & pulsating V.V.
- **Systemic signs :**

1. Tachycardia, water hummer pulse.
2. Branham's sign : slowing of heart rate as soon as the fistula is compressed.

[C] Induced : In R.F. for hemodialysis.

Investigations : 1. Doppler. 2. Arteriography.

Treatment :

[A] Congenital :

Usually no surgical treatment. If single and accessible, excision and vascular repair is done.

[B] Acquired :

- Operative interference is delayed for 3 months (to give chance for the collaterals to open).
- Excision and vascular repair, if this fails the artery is ligated above and below the fistula and its continuity is restored by graft.

Gangrene

Definition : Death (usually with putrefaction) of a macroscopic part of the body.

Etiology :

1. **Diabetic infective gangrene.**
2. **Ischemic or vascular gangrene :** (secondary to arterial obstruction).
3. **Neuropathic :** as in syringomyelia & leprosy.
4. **Traumatic :** may be direct due to crushing or pressure (bed sores), or indirect (arterial injuries).
5. **Physiochemical :** as in burns, corrosion or frost bite.
6. **Infective :**
   (a) Specific infections : Clostridial gas gangrene.
   (b) Non-specific infections : as carbuncle, cancrum oris.
7. **Venous gangrene :** due to massive thrombosis of iliofemoral vein (Phlegmasia cerulae dolens).

Clinical types :

Items	Dry gangrene	Aseptic moist gangrene	Septic moist gangrene
Tissues	Hard, dry, wrinkled and shrunken.	The part remains of the same size and consistency but discolored.	Swollen, edematous, markedly inflamed and of offensive odor.
Etiology	Gradual ischemia giving time for surface evaporation & for venous & lymphatic return.	1. Acute ischemia. 2. Artery is blocked with impairment of venous and lymphatic drainage. 3. Coincident venous occlusion.	**It may be :** 1. Primary septic gangrene : Sepsis is the cause. 2. Secondary septic gangrene : Infection of a sterile gangrene.
Color	Dark brown or black.	At first dead white, later purple or greenish black.	**C/P :** There is severe inflammatory reaction in the living tissues above it. Toxemia is severe.

Clinical picture :

[A] History of the cause.

[B] Stage of threatened gangrene : (**P**ress and **S**ee **H**ow **C**olor **F**ades)

1. Loss of **P**ulsations.
2. Loss of **S**ensation.
3. Loss of **H**eat.
4. Fixed **C**olor changes.
5. Loss of **F**unction.
6. Capillary circulation still present.

[C] Stage of gangrene :

1. General manifestations :
   - Marked in septic gangrene & mild in dry gangrene in the form of toxemia; fever, headache.
2. Local manifestations :
   - As stage of threatened gangrene.
   - **No capillary circulation.**
   - The affected part is green, brown or black due to disintegration of Hb with formation of iron sulphide.

Investigations : It differs according to the type of gangrene.

Special Varieties of Gangrene

Diabetic foot infection & gangrene

Predisposing factors :

- Diabetic patients are susceptible to serious foot infections due to :

1. Peripheral neuropathy : This makes the patient unaware of injuries insults.
2. Vascular affection : Either the major vessels are affected by atherosclerosis, which usually occurs at a young age group, or the terminal arterioles are affected by micro angiopathy.
3. Compromise of the immune system : Both humoral and cellular immunity are disturbed.

Etiology : May be due to one of the following :
1. Pure infection.
2. Pure ischemia : This is a dry ischemic gangrene occurring in a diabetic patient with major vascular obstruction.
3. Neuropathy.
4. The mixed type : There is severe infection in addition to major vascular & neuropathic affection.

Clinical picture :

[A] Infective type : Usually there is history of a trivial trauma or infection.
1. Virulent spreading infection involves the foot with severe edema.
2. Fascial spaces, tendon sheaths and the bones are rapidly involved.
3. Multiple pockets of pus develop.
4. Local : Tissues become necrotic.
5. General : patient looks very toxic with high pyrexia & may develop septicemic shock.

[B] Ischemic type : As in ischemia but at younger age and more progressive.

[C] Neuropathic type : There are large infected ulcers over pressure areas in the foot, e.g. the heel. Later severe distortion of the bones of the foot occurs with the development of Charcot's joints.

Investigations :
1. Urine for sugar, blood sugar curve & culture and sensitivity for discharge.
2. Foot X-ray for osteomyelitis
3. In case of ischemic gangrene : Duplex ultrasound&CT angiography.
4. **MRI**: to assess the condition of the soft tissues.

Treatment :

I- Prevention :

1. Proper control of diabetes.
2. Careful trimming of toenails.
3. Avoidance of walking bare footed.
4. Avoidance of tight shoes.
5. Early treatment of tinea pedis infection.
6. Daily foot care : Washing, drying, powdering & inspecting them for wounds or interdigital infection.

II- Treatment of stabilized cases :

[A] Senile type : Proper control of diabetes + Treatment of atherosclerotic ischemia.

[B] Infective diabetic type :
1. Proper control of diabetes.
2. Proper antibiotic after culture and sensitivity.
3. Excision of sloughs by experienced surgeon leaving well-drained raw area. Then frequent dressings are essential (repeated debridement and drainage of pockets is usually needed).
4. Amputation :
   (a) Minor foot amputation if there is osteomyelitis.
   (b) Urgent high amputation may be indicated.

Ischemic Gangrene (Thrombotic Gangrene)

Etiology :

1. Primary thrombosis : Rare.
2. The thrombosis is usually secondary to arterial disease such as senile atherosclerosis, diabetic angiopathy and Burger's disease

Senile gangrene :

- The commonest variety in civil practice.
- Usually confined to lower limbs. The gangrenous process is usually precipitated by slight trauma.

Clinical picture :

- **Incidence :** Usually in males, above 50 years, smoking, obese and diabetics. When it develops at an earlier age. It is generally associated with some predisposing causes such as diabetes, hypertension, hypercholesterolemia, nephritis or alcoholism.
- **Stage of ischemia, Stage of pregangrene & Stage of gangrene.** (See before)

Investigations : (see before in ischemia)

Treatment :

[A] Stage of ischemia.

[B] Established gangrene : if possible, treatment of ischemia will lower the level of amputation:

1. Care of the affected part by keeping it absolutely dry.
2. Wait for spontaneous separation in cases with limited sterile dry gangrene.
3. Amputation as before in ischemia.

Special types

Traumatic gangrene

[1] Direct traumatic gangrene e.g. bed sores (Decubitus ulcers).

- They result from prolonged pressure, which deprives the tissue of its blood supply. They occur over bony prominences such as the sacrum, ischial tuberosities, trochanters, heels or scapulae.
- **Predisposing factors :**

1. Loss of sensation as in spinal cord injuries.
2. Bad general condition.
3. Bad nursing of bed-ridden patient.

[2] Indirect traumatic gangrene is due to interference with the blood supply.

- **Frostbite :** Severe cold → freezing of tissues → thrombosis of arterioles.
- **Trench foot :** Severe cold to foot → spasm.

Buerger's disease (Thromboangitis obliterans)

Definition :

- Inflammation and thrombosis, of unknown cause, of small arteries & veins, with perivascular fibrosis which blends artery, vein and nerve into one mass causing early neuritic & severe rest pain.

Incidence : (See before, the table)

Clinical picture :

1. Buerger's disease occurs exclusively in smokers.
2. Recurrent attacks of digital ischemia *(Raynaud's phenomenon)* and superficial thrombophlebitis.
3. Chronic ischemia: as general, it is characterized by:

(1)Claudication in sole only **(2)Early severe rest pain**

4. Distal arterial occlusion, with palpable popliteal pulse.
5. Inter-digital fungus infection is a common association in Buerger's disease.
6. Angiography is ***rarely*** necessary to confirm the diagnosis.

Treatment :

1. Smoking must be stopped. **2. Sympathectomy** gives good results.

3. I.V or intra-arterial PG-E may be useful in patients with poor distal run-off.

Raynaud's disease

☒ Vasospastic disease affecting digital arteries. It affects young females in cold weather.

Possible Aetiological factors of Reynaud's disease

1. Psychological instability
2. Increased sympathetic tone.
3. Abnormal sensitivity of small arteries and arterioles of the hands to the cold.
4. Presence of cold agglutinin in the blood which cause agglutination of RBC'S on exposure to cold.

Clinical picture :

bilateral and symmetrical

1. Cold precipitates attacks of 3 phases :

(a) **Pallor**: from arteriolar spasm.

(b) **Cyanosis**: Accumulating metabolites causing congestion and cyanosis.

(c) **Rubor (red)** : As the attack ends, arterioles relax.

2. Between attacks, skin is normal.

3. The radial and ulnar pulses are preserved.

4. In late cases, trophic changes :

(a) Superficial ulcers. (b) Dry gangrene of digits tips.

Grades of Reynaud's disease

1st - Reynaud's phenomena only.

2nd - mild trophic changes in the tips of fingers and nails.

3rd - gangrene of the tips of fingers.

Treatment :

- Is only successful in early cases before arteriolar obliteration and trophic changes.

[A] Conservative for mild cases :

1. Avoiding cold. 2. Vasodilators. Ca channel antagonists may be helpful in early stages

[B] Sympathectomy : (for severe cases)

- Immediate results are good, but after few months mild sensitivity to cold returns. This partial relapse denotes that Raynaud's disease is not due to sympathetic over sensitivity but due to direct arteriolar over sensitivity to cold. Sympathectomy apparently raises the threshold at which spasm occurs.

Raynaud's Phenomenon

- Raynaud's phenomenon means similar digital vasospasm 2ry to :

[A] Certain diseases :

1. Collagen diseases e.g. lupus erythematosus, and rheumatoid arthritis.
2. Atherosclerosis.
3. Burger's disease.
4. Scalene syndrome : Such patients improve only if underlying disease can be controlled.

[B] Occupations using vibrating tools e.g typists.

- Such patients are cured by changing their occupations.

N.B.

- **Raynaud's phenomenon does not benefit from sympathectomy.**
- **Cryogloubulinemia is accompanied by Reynaud's phenomena.**
- **It may be a side effect to chronic intake of ergot containing drugs.**

N.B.: Causes of Gangrene in U.L. :

1. Thoracic outlet syndrome. 2. Arteritis. 3. Raynaud's. 4. D.M.
5. Leprosy. 6. Syringomyelia. 7. Embolism. 8. Buerger's. 9. Trauma.

Vasomotor diseases

1. Raynaud's disease & phenomena.

2. Acrocyanosis : Affects hands & feet, in female, on exposure to cold.

- **D.D. from Raynaud's by :**

(a) Extremities never completely normal. (b) No blanching.

- **Treatment :** Conservative - Sympathectomy.

3. Erythrocyanosis frigid (Bazin's disease) : Affects fatty legs in female.
Cyanosis from calf down with nodules & ulcers.

4. Erythromylagia : The warmth, the exercises & the dependency cause redness, warmth & burning pain, which are relieved by cooling.

5. Causalgia : See peripheral nerve injuries.

6. Sudeck's atrophy : See orthopedics.

Arteritis

1. Buerger's.
2. Takayasu's arteriopathy : Pulselessness diseases of female (autoimmune). Treated by cortisone.
3. Infective arteritis.
4. Endarteritis obliterans.
5. Polyarteritis nodosa.

Extracranial cerebrovascular disease

It is the third leading cause of death and is a major source of disability among elderly people.

Pathogenesis :

- Embolization is the commonest cause of TIA's and ischemic stroke.
- The embolus may arise from the heart, but more commonly it arises from an ulcerating atherosclerotic plaque at the carotid bifurcation.

Clinical features :

1. Asymptomatic patients.
2. Patients with TIA's : Carotid artery TIA's & Vertebrobasilar TIA's. (see internal medicine)
3. Patients with stroke & residual neurologic deficits.

Investigations :

1. Duplex study of the extracranial cerebrovascular arteries.
2. Arteriography is associated with a 1-2% stroke rate.
3. CT scan of the brain

Treatment :

[A] Surgery :

Carotid endarterectomy : The atherosclerotic plaque at carotid bifurcation is removed surgically.
Indications : in Internal carotid artery stenosis of 70% or greater in a patient with :
1. Carotid artery TIA's.
2. Stroke leaving minimal neurologic deficit.
3. Asymptomatic lesion.

[B] Medical treatment :

Indications :
1. Less than 50% stenosis even if symptomatic.
2. Patients during the acute phase of a stroke. Revascularization is contraindicated as it leads to hemorrhage in the infracted brain area.
3. Patients suffering from stroke with poor recovery.

Medications :
1. Antiptatelet drugs such as aspirin or ticlopidine.
2. Control risk factors.
3. Full anticoagulation in patients with cardiac embolic disease.

Subclavian steal syndrome

• In this syndrome cerebral ischemia is caused by the reversal of flow in the ipsilateral vertebral artery distal to a proximal lesion in the first part of the subclavian artery

Clinical features :

• Vertebrobasilar insufficiency occurs on exercise of the upper limb due to increased demand for blood.
• The extremity steals from the cerebral circulation through the ipsilateral vertebral artery.

Investigations : 1. Duplex scanning 2. Arteriography

Treatment :

1. Carotid-subclavian artery bypass
2. Percutaneous transluminal balloon angioplasty (PTA) of subclavian stenosis.

Endovascular surgery

• Endovascular surgery is the management of vascular diseases percutaneously through a puncture to deal with a lesion in a remote site.

Classification :

1. System : Arterial or Venous
2. Purpose : Diagnostic or Therapeutic

Therapeutic procedures :

1. Angioplasty.
2. Intravascular stents.
3. Thrombolytic therapy.
4. Atherectomy.
5. Peripheral laser angioplasty.
6. Endoluminal grafts : In Occlusive disease & Aneurysms.
7. Inferior vena cava filters.
8. Catheter vascular access.

Balloon angioplasty (percutaneous transluminal angioplasty = PTA)

Indications : These are the same as those for conventional vascular operations.
1. Critical ischemia
2. Severe claudication that interferes with the patient's work and life style.

Ideal lesion :
1. At least 5mm beyond origin of an artery.
2. Stenosis < 5cm length.
3. Best in iliac artery

Intravascular stent

Aim : may be inserted in the same session after PTA to prevent its drawbacks

Principle :

• A metal vascular stent is guided over a guide-wire, endoluminaly, and is directed to the arterial segment that has been dilated.
• The stent is carried over an expanding device that is kept closed until it reaches the desired site.
• The device is then opened to expand the stent so that it will fit in the artery

Types :
1. Balloon- expandable stents.
2. Spring-Loaded, self-expanding stents.
3. Thermal memory stents.

Inferior vena cava filter

Indications :

1. DVT or documented pulmonary embolism in a patient with a contraindication to anticoagulation.
2. Recurrent & chronic pulmonary embolism despite adequate anticoagulation.
3. Complications of anticoagulation that forced therapy to be discontinued.
4. Failure of previous caval interruption, evidenced by recurrent pulmonary embolism.

Endoluminal grafts

Principle : A combination of a stent with prosthetic graft positioned with a delivery system.
Uses :
1. Exclusion of A.A.A.
2. Support of the intima of an artery after balloon dilatation in some cases.

Venous Disorders

Venous Disorders

Superficial vein thrombosis

(Superficial thrombophlebitis)

Etiology :

1. Varicose veins.
2. Cannulation for IV infusion.
3. After injection of irritant drugs.
4. In association with :
   (a) Buerger's disease (thrombophlebitis migrans). (b) Polycythemia.
   (c) Polyarteritis. (d) Visceral cancer (may be the earliest sign of malignancy).

Clinical picture :

1. The vein becomes red, painful & cord like. 2. Pyrexia.

Complications :

Spread to deep veins via the communicating veins. In this condition proximal ligation & disconnection should be done.

Treatment :

1. Compression by elastic stocking.
2. Anti-inflammatory drugs e.g. aspirin. 3. Antibiotics (if there is evidence of infection).
4- Anticoagulant therapy is given in sever progressive cases.

Deep Venous Thrombosis (DVT)

Incidence :

The true incidence of DVT is not exactly known as many cases pass unnoticed.

Predisposing factors :

[A] Vircow's Triad :

1. Damage to the endothelial lining of the vein wall due to :

(a) Trauma to the vein wall, e.g. during pelvic operation.
(b) Inflammatory process near the vein, e.g. pelvic sepsis.

2. Venous stasis due to :

(a) Prolonged recumbency, long trips or casts.
(b) Congestive heart failure.
(c) Venous compression by tumors, pregnant uterus or pillows under the knees.

3. Hypercoagulability of blood: it may be either:

Primary hypercoagulability due to:

(a) Antithrombin III deficiency.
(b) Deficiency of proteins S & C, α2 macroglobulins, α1 antitrypsin & heparin cofactor which have an anticoagulant effect.
(c) Polycythemia and dysfibrinogenemia.
(d) Factor V leiden gene defect or activated protein c resistance.

Secondary hypercoagulability due to:

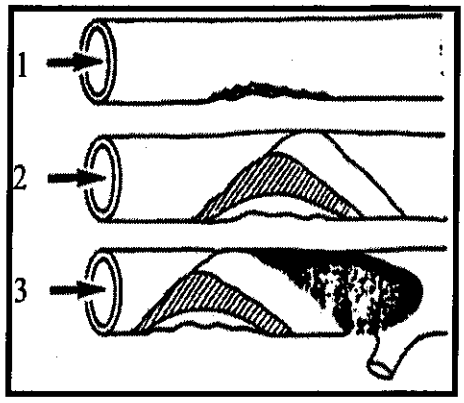
Oral contraceptives or malignancy specially ovarian cancer.

[B] Added to these factors are :

1. Fever especially typhoid.
2. **M**ajor trauma & **M**alignancy.
3. **O**besity, **O**ld age & **O**ral contraception.

Pathogenesis:

1. The process usually starts in the calf venous sinuses or in the iliofemoral veins by adherence of platelets to the endothelial surface forming a **gray cluster.**



2. Fibrin and R.B.C.'s are deposited as layers in-between the platelets giving a laminated appearance known as the **lines of Zahn.**

3. When the vein is totally occluded, a non-adherent, **jelly-like propagated thrombus** spreads up the vessel as far as the next major tributary. The thrombus is loosely attached and it can be easily detached leading to pulmonary embolism.

4. Later, the thrombus becomes tightly adherent to the vein wall producing **destruction of the valves.**

5. Later on **recanalization** of the vein occurs but the valves are permanently destroyed.

Clinical picture :

I- The Classical picture : There is triad of :

1. Pain : There is usually aching discomfort and tightness in the involved calf or thigh.

2. Swelling : It is the most reliable physical sign. It is evidenced by measuring the difference in the circumference between both sides.

The level of swelling in D.V.T differs according to the site of obstruction :

- In calf thrombosis the swelling is limited to the foot and ankle.
- In femoral thrombosis the swelling involves the calf and lower part of the thigh.
- In iliofemoral thrombosis there is massive swelling affecting the whole lower limb.

3. Tenderness : is present on compressing the muscles against the bones.

N.B.: Dorsiflexion of the foot causes pain in the calf in cases of D.V.T. (Homan's sign).
However this sign is not reliable and may cause pulmonary embolism.

II- The complication group :

1. Phlegmasia alba dolens : Massive iliofemoral DVT may be associated with severe arterial spasm, so the limb becomes pale, white & massively swollen with absent peripheral pulses.

2. Phlegmasia cerulae dolens : Massive iliofemoral DVT may be associated with severe congestion and cyanosis and the whole lower limb looks massively swollen and blue and if not properly treated it may lead to venous gangrene.

3. Venous gangrene.

4. Pulmonary embolism : manifested by severe chest pain, dyspnea & hemoptysis. (Discuss).

5. Postphlebitic limb. (see varicose veins).

Phlegmasia alba dolens :	means painful white swelling.
Phlegmasia cerulae dolens :	means painful blue swelling.

III- The asymptomatic group :

This is not uncommon. There are no local symptoms and the patient may present later with either pulmonary embolism or postphlebitic limb. However, it may be suspected by the presence of unexplained fever or tachycardia.

Investigations :

As clinical examination is not reliable and incorrect in about 50% of cases. Investigations should be done before starting treatment.

[1] Laboratory tests: D-dimer:

This is a degradation product of fibrin. It is elevated with DVT and PE. A normal value excludes pulmonary embolism.

[2] Doppler ultrasonography :

- Normally upon applying the ultrasound probe over a patent vein, a continuous sound is heard (venous hum). If the probe is applied over the femoral vein & pressure is applied to the calf, the hum gets changed into a roar due to increased blood flow.
- If there is thrombosis in the popliteal or femoral veins, the roar doesn't occur.

Advantages : It is a simple & rapid method & is accurate in 80-85% of cases.
Disadvantages : insensitive in calf vein thrombosis.

[3] Duplex ultrasound :

Advantages :
(a) This method combines doppler ultrasound flow analysis with ultrasound imaging.
(b) Detects partially occlusive thrombi.
(c) Its sensitivity & specificity reach 90-100% & most duplex errors are in below knee veins. **So this test is the standard test for diagnosis of DVT and its accuracy approaches that of venography.**

Doppler & duplex scan findings in case of DVT :

Items	Normal	DVT
Vein diameter	Normal	Dilated vein
Blood flow	Spontaneous	Poor
Echogenic material in lumen	None	Present
Distal compression	Augments blood flow	Poor augmentation
Blood flow with respiration	Phasic flow with respiration	Loss of phasic flow with respiration

[4] Enhanced helical computed tomography (CT) : is the most recent diagnostic test. It shows the thrombus even in small veins.

N.B 1: ^{125}I-fibrinogen uptake: and ascending venography are no more used for the diagnosis of DVT.
N.B 2: Other tests: as plethysmography & venous pressure measurements are of academic interest.

Differential diagnosis :

1. **Contusion of Calf muscles.**
2. **Rupture of plantaris tendon :** Venography & duplex may be needed to establish the diagnosis.
3. **Lymphatic obstruction :** The swelling is chronic and usually non-pitting.
4. **Cellulitis :** There are systemic & local symptoms & signs of inflammation. The leg is hot & red.
5. **Acute arterial occlusion :** There is more pain, no swelling, diminished sensations, lost pulse.
6. **Ruptured Baker's cyst.**

Treatment :

I- Prophylactic :

[A] Physical measures to reduce venous stasis. These include :

1. Early ambulation after operations. 2. Active leg exercises.
3. Elastic stocking support 4. Adequate postoperative hydration.
5. Intermittent pneumatic external calf compression by special devices.
6. Elevation of the foot of the bed.

[B] Prophylactic anticoagulation : In high-risk patients (lower the risk by 50%):

1. Low dose heparin (Mini Heparin) : 5000 IU subcutaneous2 hours before operation and then every 12 hours until the patient is ambulant.

- **Prophylactic heparinization** should not be used if a large raw area is left after surgery.

2. Low molecular weight dextran : 500 ml IV/12 hours.

N.B: prophylactic anticoagulant is indicated for major surgeries, History D.V.T or Pulmonary embolism, Contraceptive pills taken by a female, Obesity and obesity

II- Curative : The objectives of treatment are :

1. Prevention of formation of new thrombi.
2. Prevention of pulmonary embolization.
3. To minimize venous valves damage.

[A] Bed rest and elevation of the lower limb :

- **12-15 degrees above the level of the heart :** Elevation reduces edema and pain, increases venous return and also can prevent further thrombosis
- **Application of an elastic bandage or stocking :** will also help venous return. Thrombi usually take 7-10 days to become adherent to the vein wall, the patient should be kept in bed for this period.
- **Graduated ambulation with elastic support :** is then allowed.

[B] Anticoagulant therapy :

I- Heparin :

- **Action :**

1. Enhances the activity of antithrombin III.
2. Neutralizes factors IX, X, and XI.
3. Mild activation of fibrinolysis.

> Heparin is given until all signs of active thrombosis subside. This usually takes 7-10 days.

There are 2 types of heparin:

- ***Low molecular weight heparin(fractionated heparin)***

Tenzoparin: 175 IU/day SC.
Enoxyparin: 1 mg/Kg /12 hours SC.

- ***Unfractionated heparin: can be prescribed in one of 3 methods :***

1. Continuous IV infusion: A bolus dose of 80 IU/Kg is given IV and then dextrose 5% drip containing heparin is infused at a rate of 18 IU of heparin/kg/hour. *The ideal method*
2. Bolus therapy: 70-80 IU/Kg intravenously every 4-6 hours.

- **The dose is monitored by :**

1. Measuring the **Activated Partial Thromboplastic Time (APTT).**
   > This should be kept between g1.5 - 2.5 times the control values.
2. A more practical but less accurate method is to do **Clotting time.**

- **Complications of heparin therapy :**

1. Bleeding due to over dosage. It can be treated by giving protamine sulphhate 1 mg IV for every 100 IU of heparin.

2. Failure to respond to heparin (heparin resistance): It may be mild and requires only increasing the dose of heparin.

3. Heparin induced thrombocytopenia: occurs usually in the second week, heparin should be stopped.

	Unfractionated heparin	LMWH
Mode of action	Antithrombin III	Anti Xa
Half-life	1.5-2 hours	12 hours
Route of administration	IV	subcutaneous
Monitoring	Activated partial thromboplastin time	Not required-if needed: level of Xa
Incidence of heparin induced thrombocytopenia	1-5 %	Less than 2% *Less likely to cause bleeding*
	The patient should be hospitalized	May be taken at home.

II- Oral anticoagulants (coumarin derivatives) : The most commonly used is **(Warfarin)**

- **Action :**

Blocks the synthesis of at least 4 vitamin K dependent cloning factors (prothrombin and factors VII, IX and X), for this reason its anticoagulant effect is delayed.

- **Method and dose of administration :**

An initial dose of 10 mg warfarin is followed by 5 mg daily dose.
Heparin therapy must be continued in the first 3 days of treatment.
Oral anticoagulants are given for 3 - 6 months, which is the time needed for recanalization and collateralization.
In some patients who are liable to rethrombosis, warfarin is given indefinitely.

- **The dose is monitored by :**

The level of prothrombin concentration : aiming to be 30 - 40% of control.

- ✓ PC should be measured before the start, 5 days after then every 2 weeks repeatedly.

- **Complications of oral anticoagulants :**

1. Bleeding : is the main problem and is treated by vitamin K IV injection.
In rare cases severe shock may occur form massive retroperitoneal hemorrhage. It is treated by Lower ing the dose but in severe cases by fresh blood or fresh frozen plasma transfusion and vitamin K injection.
2. Interaction between oral anticoagulants and other drugs : like aspirin, H2 receptor blockers and non-steroidal anti-inflammatory agents leading to an increase in the anticoagulant effect.

Contraindications to anticoagulant therapy :

[A] Absolute :

1. Bleeding tendency. 2. Septic phlebitis. 3. Bacterial endocarditis.
4. Trauma to or recent operation on the brain or spinal cord.

[B] Relative :

1. Major visceral injury. 2. Major acute fractures.
3. History of cerebral hemorrhage. 4. Peptic ulcer.
5. Hypertension where the blood pressure is higher than 180/120 mmHg.

[C] Thrombolytic therapy :

Action :

Fibrinolytic activators lyse fresh thrombi and may preserve the competence of venous valves.
Examples of fibrinolytic activators include : streptokinase, urokinase & tissue plasminogen activator (TPA).

Complications :

1. Allergic reactions.
2. Severe bleeding.
3. They are very expensive.

[D] Operative treatment :

In a small percentage of cases operation is necessary.

1. Venous thrombectomy : using **Fogarty's venous catheter.**
Construction of a temporary small AV fistula may improve its results.

Indication of venous thrombectomy :

1. Massive iliofemoral thrombosis.
2. Phlegmasia cerulae dolens.
3. Pulmonary embolism.

2. Venous interruption :

Method :

Insertion of venacaval filter (Greenfield) usually in an Infrarenal position opposite to the 2nd lumbar vertebra.

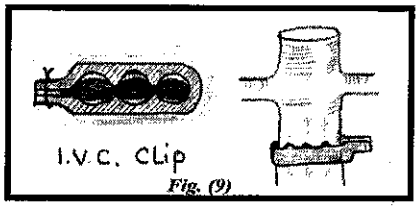


Fig. (9)

Indications :

Cases with repeated showers of pulmonary emboli that do not respond to medical treatment.

Complications: (rare)
Hemorrhage, injury of IVC, perforation of aorta, migration.

Surgical Physiology

Factors that help the venous return from the lower limbs :

- The venous drainage of L.L. is carried out against gravity (from distal to proximal and from superficial to deep veins) by :

1. Muscle pump which needs strong muscles and intact deep fascia :
When the muscles contract within a tight deep fascia, deep veins are compressed → blood is directed towards the heart by competent valves. When muscles relax, pressure drops in the emptied deep veins → blood is sucked from the superficial veins.

2. The transmitted arterial pulsations to the venae commitants.

3. The unidirectional valves.

4. The negative intrathoracic pressure : Pressure in the veins on dorsum of foot falls during walking to 30 cm H2O from the resting venous pressure of 100 - 120 cm H2O.

N.B1: the perforating veins

a) Indirect perforators: pass from the superficial viens to the soleal plexus and then to the deep veins
b) Direct perforators: pass from the posterior arch (branch of the long saphenous vein) to the posterior tibial vein.

N.B 2: the venae cavae are exceptions as they have no valves.

Varicose Veins Of The Lower Limbs

Definition :

Dilated, elongated, tortuous superficial veins of the L.L.

Etiology & Types :

Primary V.V	Secondary V.V
Two theories : 1. Congenital weakness of the wall of veins → venous dilatation → valvular incompetence. 2. Congenital valvular incompetence. **Aggravating factors :** 1. Commoner in females. 2. High parity. 3. Occupations requiring prolonged standing. 4. Constricting clothes.	**Secondary to one of the following :** 1. Deep vein thrombosis (the commonest cause). 2. Arteriovenous fistula which may be : (a) Congenital. (b) Acquired due to : Trauma or Shunting for dialysis purposes → high venous pressure. 3. Compression of the deep veins by : Tumors or Pregnancy.

➧ **Klippel-Trenaunay syndrome (KTS) is a rare congenital vascular disorder in which a limb may be affected by secondary varicose veins.**

Gross picture (pattern) :

1. Tubular type : dilated main veins (common in primary V.V).
2. Spider type : veins are thin and blue (dilated skin venules).
3. Serpentine : veins are coiled.
4. Saccular type : Blow out at the site of incompetent perforator with a palpable defect in the deep fascia opposite it.

- **Saphena Varix** is a saccule opposite an incompetent saphenofemoral junction.

Complications :

Complications are much more frequent with secondary varicose veins. **They include :**
1. Edema.
2. Subcutaneous bruises : due to rupture of small veins as a result of venous hypertension.

3. Itching, dermatitis & eczema : are due to deposition of hemosiderin in subcutaneous tissues.
4. Liposclerosis : The extravasated fibrinogen leads to fibrous tissue formation. The soft subcutaneous fat is replaced by tough fibrous tissue.

5. Recurrent superficial thrombophlebitis.
6. Venous ulceration.

- The combination of the above mentioned complications, together with chronic leg pain constitute the **post-phlebitic syndrome** as they usually follow previous deep vein thrombosis with incompetent perforating veins.

7. Hemorrhage : may occur due to rupture of varicose veins.

- **Long Saphenous vein** is the longest vein in the body and the most commonly affected by V.V.
- **V.V. is commonest in lower limb. Why?**

1. Marked length of veins.
2. Vertical position.
3. Large volume of blood they contain.
4. The liability of compression by abdominal mass (e.g. uterus).

Clinical Picture :

Primary varicose veins	Secondary varicose veins
Patient	
1. Occupation with prolonged standing or sitting. 2. Positive family history in 50% of cases.	1. Females with repeated pregnancy, abortion, prolonged intake of oral contraceptives. 2. Patients with dehydration (prolonged vomiting or diarrhea), prolonged recumbency or prolonged fever.

Symptoms	
[1] Cosmetic disfigurement.	
[2] Pain (venous pain) :	
Mild	Marked
It is dull aching, heaviness or bursting in character, increased by prolonged standing or sitting and relieved by elevation of the L.L. and walking. **Walking aggravates pain if there is :** 1. Incompetent perforators → high venous pressure on walking (up to 150 mmHg). 2. Associated ischemia. 3. Active D.V.T. with occluded deep veins.	
[3] Night cramps.	
[4] Swelling (edema) :	
Slight ankle swelling that resolves after night sleep.	Persistent diffuse swelling of the L.L does not resolve by night sleep.
[5] Skin Complications :	
Less common.	More common.
Past History	
• Nothing related.	• D.V.T. • Stab or bullet (A-V fistula).
Signs	
[A] General Examination :	
There may be flat foot, hernias, kyphosis, varicocele or piles (congenital mesenchymal weakness).	• Pelvic or abdominal tumor, pregnancy. • Scar of pelvic operation. • Pulse & B.P. changes in A - V fistula.
[B] Local Examination :	
Inspection :	
1. The V.V. are multiple, dilated, elongated, tortuous, bluish swellings along the course of veins of L.L.	
2. Usually bilateral unequal.	2. Usually unilateral.
3. Affect long or short Saphenous veins or the communicators.	3. Affect any vein or venule in an irregular distribution and may extend to the abdomen.
4. V.V. never crosses the groin.	4. V.V. crosses the groin in cases of iliofemoral or I.V.C. obstruction.
5. The veins are mainly tubular.	5. Mainly serpentine or spider-like.
6. Minimal pitting ankle edema may be present.	6. Diffuse massive edema of L.L., which becomes hard & non-pitting in chronic cases.
7. Skin complications are less common.	7. Skin complications are more common.

Palpation :	
1. Dilated, elongated, tortuous, soft, compressible tubules. 2. While the patient is lying down, a defect is felt in the deep fascia opposite each blow out (incompetent perforator) → **Fegan's method.** 3. Incompetent saphenofemoral or saphenopopliteal junction shows expansile impulse and thrill on cough. 4. Saphena varix is a soft cystic compressible swelling with impulse and thrill on cough. 5. Palpate the V.V. for firm tender nodules (superficial thrombophlebitis).	
6. No spontaneous pulsation, thrill, murmur or local gigantism.	6. Spontaneous pulsations, murmur and thrill & local gigantism in case of congenital A-V fistula.

- **Special tests :** See clinical notes.

Investigations :

- The aim is detection of the sites of incompetent communicating veins and to verify the patency of the deep veins. It should be noted that most cases of primary varicose veins require no investigations because careful clinical assessment provides enough information.

1. Duplex ultrasound imaging.

2. Venography :

Values : It shows :

- Patency and size of deep veins.
- Presence of valves and any incompetent perforator.

Method :

- A tourniquet is applied above the malleoli to occlude the superficial veins.
- Inject the contrast material into a superficial V. on the dorsum of foot and serial X-ray films are taken.

3. In case of varicose ulcer :

- Plain X-ray (if periostitis is suspected) and biopsy (if malignancy is suspected).

Differential diagnosis :

1. Other causes of leg pain.
2. Other causes of leg swelling.
3. Other causes of leg ulcers.

Treatment :

[1] Conservative treatment : For

(a) Secondary varicose veins : If deep veins are occluded, the only treatment is conservative. If deep veins are patent (canalized deep vein thrombosis), still conservative treatment is essential, but injection or operation can be added if varicose veins are large or complicated.

(b) Primary varicose veins : If very early or patient is pregnant, unfit for, waiting for or refusing operation. Conservative treatment includes :

1. **Below knee elastic stocking or crepe bandage** to support superficial veins, to prevent reflux from deep veins thus helping leg muscle pump and to prevent edema.
2. **Periodic leg elevation** to prevent stasis and congestion.
3. **Regular exercises** to restore muscle tone.
4. **Long sitting or standing is avoided, but walking is encouraged.**

[2] Injection treatment : (Compression sclerotherapy)

Indication :

1. Early cases of 1ry V.V., which come for cosmetic reasons.
2. Residual V.V. after operation.
3. In secondary varicose veins, it can be done if deep veins are patent.

Methods of injection compression sclerotherapy :

Aim :

- Sclerosants like 5% ethanolamine oleate or 3% sodium tetradecyl sulphate if injected in an empty vein leads to injury of the endothelial layer of the intima. If compression is then applied, the two walls of the vein will adhere together leading to **obliteration of the vein by fibrosis and not by thrombosis.**
- If injection is done with the vein full of blood, thrombosis will occur which later recanalizes with recurrence of varicosities.

Technique :

1. A venous tourniquet is applied over the thigh while the patient is standing and the site of the vein is identified and marked on the skin by a skin marker.
2. The patient is asked to lie down and a thin hypodermic needle is inserted into the vein.
3. An assistant then elevates the limb and 1ml of the sclerosant material is injected.
4. Immediately after application of bandage, the patient is instructed to walk for a long distance in order to flush any amount of sclerosant that might have reached the deep veins.

Complications :

1. Extravasation leads to sloughing of the skin and poor cosmetic results.
2. Deep venous thrombosis may occur if a large amount of the sclerosant material reaches the deep system undiluted. So, No more than 1 ml is injected at any point and No injection is done for veins above the knee.

[3] Surgical treatment : (see operative notes)

Indications :

1. Large 1ry V.V.
2. Incompetent valves of long Saphenous or short Saphenous.
3. Presence of complications.
4. Cosmetic reasons.
5. In secondary varicose veins, it can be done if deep veins are patent (canalized deep vein thrombosis), but just in addition to conservative treatment.

Management of complications : e.g.:

1. Varicose ulcer : see later.
2. Hemorrhage : Elevation and pressure bandage.

Varicose (Venous) Ulcer

Venous ulcers constitute 90% of chronic leg ulcers

Definition :

It is chronic leg ulcer complicating 2ry V.V. usually in post-phlebitic limb, much less common with A-V fistula.

It is due to : incompetent ankle perforators causing reflux of blood from deep to superficial veins with consequent maximal congestion leading to anoxia. Thus skin vitality at ankle is reduced. Ulceration usually occurs above medial malleolus **(ulcer bearing area).** If it heals, recurrence is common until something is done to correct reflux and venous hypertension at ankle. Excess fibrosis makes its base and edge hard (callous) and such ulcer resists healing (indolent).

N.B.Rarely the venous ulcer results from 1ry V.V.s. In this case it is usually transient and heals rapidly

Clinical picture : Postphlebitic syndrome.

Characters of venous ulcer :

Site : At medial malleolus **(ulcer bearing area)** as the skin of the lower part of the leg is drained directly by the perforators, which drain into the deep system.
Margin : shows varicosities, edema, brown pigmentation, eczema and itching marks.
Base : is tender, soft if recent and hard if chronic, may become fixed to tibia, and underlying periosteitis may produce thick tender bone.
Limb examination : reveals all manifestations of postphlebitic syndrome and firm tender inguinal nodes.

The Ulcer is chronic due to :

Tissue anoxia : due to venous stasis and impaired nutrition leading to liberation of free oxygen radicals which are toxic to the tissues.
Chronic irritation by hemosiderin (eczema).
Superadded infection.
Superadded periosteitis.
Liposclerosis : the fibrin acts as a barrier to O2 and nutrients.

D.D.: Other chronic leg ulcers.

Complications : (as any chronic ulcer)
1. Malignancy : Marjolin ulcer.
2. Periosteitis : Suspected if ulcer is fixed to tibia, which is thick and tender.
3. Talipes Equinus : May complicate varicose ulcer as walking on toes decreases the pain.
- **Treatment :** Physiotherapy.

Investigations : For suspected complications (not routine)
1. Biopsy : for suspected malignancy.
2. X-ray : for suspected periosteitis.
3. Venography : if surgery is indicated.

Treatment :

[A] Conservative : For all cases.
1. If very small uninfected and not associated with severe edema, they can heal with ambulant conservative measures.
2. If large or infected or associated with severe edema or ambulant treatment failed, patient must be hospitalized for bed rest.

- **It includes :**

(a) Elevation of the foot in bed.

(b) Ulcer care: Moist saline dressings. The use of topical antibiotics aggravates the condition by inducing allergic reaction.

(c) Elastic stocking or crepe bandage.

(d) Some medications may accelerate healing e.g.:

- Pentoxiphylline (trental)
- Prostaglandin E1

- **Conservative treatment is usually successful in allowing the ulcer to heal, however, once the patients return to normal activity, most ulcers will recur.**

[B] Surgery :

1. If the problem is just incompetent perforators, they can be either injected or excised.

N.B: subfascial ligation of these perforators **(Cockett's operation)** and Endoscopic **subfascial ligation are not commonly performed nowadays.**

2. Covering ulcer by split thickness (Thiersch graft) or by cross leg flap : In callous ulcer, excision is done before putting graft.

- **N.B.** After treatment, patient will use elastic stocking indefinitely.

[C] Treatment of Complications :

1. Malignancy : Excision with safety margin or amputation.
2. Periosteitis : Saucerization.

Chronic Leg Ulcers

Classification : (General)

[A] Non-specific ulcers :

1. Traumatic : Due to direct destruction of surface epithelium by mechanical, thermal or chemical trauma.
2. Infective : e.g. pyogenic.
3. Vascular : Due to arterial obstruction (ischemic) or due to venous cause (varicose ulcer).
4. Trophic : Due to loss of vasomotor supply (due to destruction of local nerve supply).

[B] Specific : T.B., syphilis.

[C] Malignant : Epithelioma, fungating sarcoma.

[1] Traumatic Ulcer :

It is due to direct destruction of surface epithelium by trauma with secondary infection on top.

- **At first** the ulcer is acute and painful with congested edge and discharge is purulent.
- **In the healing stage,** the floor is covered by either healthy or unhealthy granulation tissue and edge is sloping.
- **If the ulcer becomes chronic,** the base becomes indurated with pale granulation, the discharge is sero-purulant and the ulcer becomes fixed.

Healthy granulations	Unhealthy granulations
Red	Pale
Finely granular flat surface	Coarsely granular raised surface
Does not blood easily	Bleeds easily
Minimal serous discharge	Profuse pus discharge
Painless ulcer	Painful ulcer

- **Treatment :**

1. Rest, elevation, dressings and antibiotics.
2. Grafting may be needed.

[2] Varicose Ulcer. (see before)

[3] Arterial (Ischemic) Ulcer. (see before)

[4] Trophic Ulcer :

It occurs in an area deprived of its nerve supply due to C.N.S. disease or peripheral nerve lesion. The foot is anesthetized, and the patient is unaware of trauma causing ulceration.

- **Site :** Opposite the head of first or 2nd metatarsal bone or opposite the heal.
- **Size :** Variable but it may penetrate deeply or even perforate the foot to appear on the dorsum changing into perforating ulcer.
- **Margin :** Skin cornified forming a callosity. Skin is insensitive to pain
- **Treatment :**

1. Conservative : rest, elevation and dressing.
2. Excision of the callosity track, and base.
3. Amputation if many tarsal bones and joints are diseased.

[5] Syphilitic Ulcer :

- **Site :** Upper outer part of leg (opposite head of fibula).
- **Base :** indurated. Characteristically it is painless.

[6] T.B. Ulcer :

Usually secondary to T.B. osteomyelitis or arthritis. Ulcer is shallow with undermined edge, thin cyanotic margin, soft base, pale anemic granulation tissue at the floor.

[7] Malignant Ulcer :

1. Epithelioma either arising de novo or on top of chronic ulcer.
2. Malignant melanoma.
3. Fungating sarcoma.

[8] Lymphedema Ulcer. (see later)

[9] Blood Diseases : e.g. Sickle cell anemia.

Edges of ulcers :

1. Rolled edge : because the edge of the ulcers growing slowly with attempt of healing.
2. Everted edge : because the edges of the ulcers growing rapidly.
3. Undermined edge : because infection affects subcutaneous layer more than the skin.
4. Punched out edge : because of rapid death with no attempts of healing.
5. Slopping edge : attempts of healing.

N.B:CEAP classification of varicose viens:

C stands for clinical classification:

C0 no visible or palpable signs of venous disease.
C1 telangiectasis or reticular vein .veins less than 3 mm.
C3 edema
C4 skin and subcutaneous changes
C5 healed venous ulcer
C6 active venous ulcer
Further descriptors (a) for asymptomatic, (s) for symptomatic.

E stands for etiology

Ec: congenital
Ep: primary
Es: secondary.
En: no venous cause

A stands for anatomy

As: superficial veins:
Ap: perforator veins.
Ad: deep veins.
An: no location identified.

P stands for pathophysiology

Pr: reflux
Po: obstruction
P r o: reflux and obstruction
Pn: no pathophysiology identified.

Axillary-Subclavain DVT (3% Of All Cases Of DVT)

Aetiology

1. It may be **primary**. It is usually proceeded by unusual muscle activity of the upper arm.
2. It may be **secondary** to central venous catheter, IV chemotherapy given in arm veins or metastatic tumor in axilla.

Clinical picture

1. Pain & swelling in the arm, also it may be cyanosed.
2. Fullness of the infraclavicular fossa.
3. Presence of prominent venous collaterals over the shoulder and anterior chest wall.

Lymphatic Disorders

Lymphatic Disorders

Acute Lymphangitis

Clinical Picture :

Fever, rigors and general constitutional disturbances may be very severe. Locally, there is pain, edema and red tender streaks. The regional lymph nodes are enlarged and tender.

Treatment :

1. Treatment of the cause.
2. Antibiotics, especially penicillin and broad-spectrum antibiotics.
3. Local rest of the affected part, and local heat to help resolution.
4. If suppuration occurs, it needs an incision.

Lymphadenopathy

The causes of lymph node enlargement are:

1. Inflammatory :

[A] Acute non specific :

(i) Septic lymphadenitis. (ii) Infectious mononucleosis.

[B] Chronic :

(i) Non-specific.

(ii) Specific :

- Bacterial : T.B., $.
- Viral : Lymphogranuloma inguinale, cat scratch disease, AIDS.
- Parasitic : Filarial.
- Protozoal : Toxoplasma.

2. Lymphomas :

(a) Hodgkin's lymphoma. (b) Non-Hodgkin's lymphoma.

3. Blood Diseases :

(a) Acute leukemia. (b) Chronic myeloid leukemia. (c) Chronic lymphatic leukemia.

4. Lipoidosis.

5. Metastases.

6. Collagen diseases.

Acute Septic Lymphadenitis

Clinical picture :

- The picture of the causative lesion.
- General constitutional manifestations.
- **Locally the nodes are enlarged, red, hot, tender, firm or soft and if suppuration occurs fluctuation will be evident.**

Treatment :

- Treatment of the causative focus.
- General rest and antibiotics.
- If an abscess forms, incision & drainage.

Chronic Non-Specific Lymphadenitis

Clinical picture:

- Common in the submandibular and inguinal lymph nodes.
- **The nodes are slightly enlarged, mobile, slightly tender and firm in consistency.**

Treatment:

- This is directed to the original focus. The nodes need no treatment.
- If a case of chronic nonspecific lymphadenitis persists for more than 3 or 4 months, tuberculosis must be excluded.

Tuberculous Lymphadenitis

Incidence:

- Usually affects children & young adults.

Etiology:

- **Predisposing factors :** Poor general resistance.
- **Organism :** Mycobacterium tuberculosis.
- **Pathological Types :** 1. Lymph born type : The commonest. 2. Blood born type.

Pathology:

[A] Gross picture :

1. Lymph born (fibrocaseous) type	2. Blood born (lymphadenoid) type
1. Primary infection.	1. Secondary to active T.B. focus in the body.
2. Affect a localized group of L.N.s.	2. Affect multiple groups of L.N.s
3. Initial lesion occurs in the cortex → T.B. periadenitis → matting.	3. Initial lesion occurs in the medulla → No Periadenitis → No matting
4. Caseation → cold abscess → T.B. sinus.	4. No caseation. No cold abscess.

[B] Microscopic picture : Multiple tubercles each consists of :

1. Central zone : Structureless eosinophilic caseous material.

2. Mid zone : Epitheloid cells and Langhan's giant cells (Peripheral multinuclei arranged in horse-shoe manner).

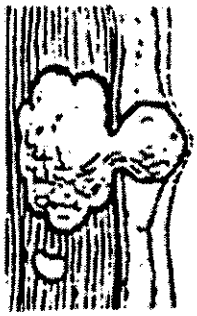
3. Peripheral zone : Small rounded cells.

Complications:

1. Cold abscess formation : C/P :

- Soft, fluctuant swelling. The overlying skin is at first normal, and then it becomes dusky (not red or edematous as in an ordinary pyogenic abscess). A cold abscess is slightly warm and slightly tender.
- The skin then thins out before rupture, which ends in a **sinus.**
- The cold abscess is actually **"not cold and not an abscess".**

> It is not cold because clinically it is warm, but is in fact colder than a pyogenic abscess.
> It is not an abscess because the contents are not pus, but caseating material.



2. Collar and stud abscess (bilocular abscess).
3. T.B. sinus.
4. Secondary infection.
5. Spread to other groups of L.N.s or miliary T.B.
6. Calcification.

Clinical picture:

1. Lymph born (fibrocaseous) type		2. Blood born (lymphadenoid) type	
• Common.	• Usually in children.	• Rare.	• Usually in old.
Local : **1. Localized lymphadenopathy** affecting the upper deep cervical L.N.s **2. Variable consistency :** Early: firm; Caseation: cystic; Calcification: hard **3. L.N.s are matted together :** L.N.s may be arranged in beaded cords due to thickening of connecting lymphatics.		**Local :** **1. Generalized lymphadenopathy.** **2. Firm in consistency.** **3. L.N.s are discrete.** • As the patient is usually an adult of fair resistance, there is no breaking down, caseation or cold abscess formation.	

N.B. The commonest groups of LNs affected by lymph born TB lymphadenitis are cervical LNs.
N.B. Blood born TB lymphadenitis may be mistaken for hodgkin's disease.

Investigations:

[A] Laboratory :

1. **CBC, ESR.**
2. **Tuberculin test** serves as a good negative test.
3. **Aspiration** of a cold abscess, and examination of the aspirate microscopically & guinea pig inoculation.
4. **Smears** from a sinus and examination for tuberculous organisms.

[B] Radiological :

- **X-ray** to the chest and mediastinum.

[C] Surgical :

- **Biopsy** from the nodes will establish the diagnosis.

Treatment:

[A] Tuberculous lymphadenitis before caseation :

1. **Antituberculous treatment.**
2. **Surgical excision** is indicated for a single group of lymph nodes showing no response to medical treatment after a period of 6 months.

[B] Cold abscess :

1. **Antituberculous drugs.**
2. **Aspiration and injection of streptomycin solution.**
   - The rules of aspiration of a cold abscess should be followed to avoid sinus formation :

(a) Wide-bored needle.
(b) The needle is inserted in a healthy part of the skin away from the abscess.
(c) The site of puncture should be in a non-dependent part.
(d) The needle should also pass in a **Valvular manner (i.e The points of entry through the skin and the abscess cavity should not be opposite to each other).**

- The aspiration usually needs repetition every few days until the abscess dries up.

3. **Incision :** Indicated in the following conditions :

(a) Secondary infection.
(b) If the abscess is imminent to rupture.

[C] Treatment of a tuberculous sinus :

1. Antituberculous treatment.
2. Dressing with streptomycin.
3. Excision with the underlying nodes, if resistant to conservative measures.

Syphilitic Lymphadenitis

Primary stage :

- The draining lymph nodes of the primary chancre are usually enlarged, rubbery, discrete, do not break down. Not painful or tender.

Secondary stage :

- Generalized lymphadenopathy.
- L.N.s are discrete, not tender and rubbery.
- The characteristic sites are epitrochlear region and along the border of the sternomastoid.

Tertiary Stage :

- There is no gumma of a lymph node (or is at least extremely rare).

Lymphomas

Definition :

- Malignant neoplasm that arises in lymph nodes or extra nodal lymphoid tissue.

The main types are :

1. Hodgkin's disease. 2. Non-Hodgkin's lymphoma. 3. Burkitt's lymphoma.

I- Hodgkin's disease	II- Non-Hodgkin's lymphoma
More common	Less common
Pathology :	
Macroscopically : • It usually starts in the cervical lymph nodes. • The affected nodes are enlarged, discrete, rubbery & have a pink color. • Usually there is a large lymph node in the center with smaller lymph nodes around it. • There may be extra nodal affection of the spleen, liver or bone marrow.	**Macroscopically :** **Lymph Nodes :** 1. Markedly enlarged, amalgamated. 2. Early invasion of the surrounding structures. 3. Variable consistency but usually hard.
Microscopically : 1. Pleomorphism. 2. The characteristic **Reed Sternberg cells.** These are giant cells that have an even number of nuclei (2 or 4), which are arranged in a mirror image manner. *Characteristic*	
Classification :	
Histological types : • In descending order of prognosis : 1. Lymphocyte predominance. *Best prognosis* 2. Nodular sclerosis. (The commonest variety) 3. Mixed cellularity. 4. Lymphocyte depletion.	• **Based on the cell of origin :** **1. B Cell lymphoma is further classified into :** (a) Small cell lymphoma. (b) Large cell lymphoma. (c) Mixed small and larger cell lymphoma. (d) Immunoplastic lymphoma. **2. T cell lymphoma.** **3. Lymphoblastic lymphoma.** **4. Histiocytic lymphoma.** • In each type the cells may be arranged in a nodular or a diffuse pattern.

Staging:

Ann Arbor staging system :

Stage I : Single involved lymph node group **(I),** or a single extra lymphatic site **(I^E).**

Stage II : Two or more involved lymph node groups limited to one side of the diaphragm or a solitary extra lymphatic site and one or more lymph node areas on the same side of the diaphragm **(II^E).**

Stage III : Involvement of both sides of the diaphragm.

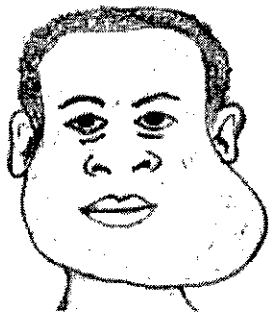
Stage IV : Extra lymphatic spread including liver, lung, bone marrow, skin and gut.

All stages are subdivided into either :

1. No systemic symptoms.
2. One or more of the three systemic symptoms; fever, night sweats, and weight loss of more than 10% in six months.

I- Hodgkin's disease	II- Non-Hodgkin's lymphoma
Clinical Picture :	
Incidence : • **Race :** More in Caucasians. • **Age :** Two age peaks, between 15-35 years, and above 50 years.	**Incidence :** More common with : 1. Sjogren's disease. 2. Immune deficiency. 3. Systemic lupus erythematosus.
• **Painless progressive enlargement of the cervical lymph modes** • With progress of the disease, other lymph node groups are affected. These nodes are **enlarged, discrete, non-tender & rubbery in consistency.** • **Splenomegaly(Toffee-almond appearance) & hepatomegaly** may be present • Some patients exhibit **systemic manifestations** in the form of fever, night sweats, weight loss, pruritis, anemia, and jaundice. • Sometimes **a characteristic intermittent fever,** which lasts for 2–3 weeks followed by a remission for few weeks, may occur **(Pel-Ebstein fever).**	• **Lymph node enlargement :** Progression of lymph node enlargement does not follow an orderly anatomical pattern as in Hodgkin's disease. • **NHL is more likely extra nodal than Hodgkin's lymphoma e.g.:** 1. Gastric lymphoma produces manifestations that are similar to carcinoma. 2. Intestinal lymphomas may produce intestinal obstruction, bleeding, or perforation. • **The disease is likely to be disseminated at the time of presentation.**
Investigations :	
[A] Laboratory : **1. Blood picture :** May show : Anemia, eosinophilia or lymphopenia. **2. High ESR.** **3. Alkaline pbosphatase :** is elevated in cases with bone or liver involvement. **[B] Radiological investigation :** **1. Chest X-ray and CT scan :** allow detection of intrathoracic disease. **2. Abdominal US and CT scan :** have replaced lymphangiography for detection of para-aortic node. **[C] Surgical investigation :** **I- Lymph node biopsy :** The corner stone of diagnosis. **II- Staging laparotomy :** • **Indications :** Stages IA, IB, and IIA • **The operation includes :** **1. Splenectomy :** For staging & avoids the need of its irradiation. **2. Biopsy of both liver lobes.** **3. Biopsy of all intra-abdominal lymph node groups,** which are marked by metal clips to help future localization by the radiotherapist. **4. Bone marrow biopsy** from the iliac crest. **5. In females** the ovaries and Fallopian tubes are fixed in the middle line behind the uterus to guard them against irradiation. • **Staging laparotomy is not preferred in many centers because :** 1. The high accuracy of CT scan, and the increasing availability of MRI. 2. The risk of overwhelming post-splenectomy infection (OPSI).	
Treatment :	
• Stage IA, IB & IIA are treated by radiotherapy. • Stage IIB is treated by radiotherapy and 6 cycles of combination chemotherapy. • Stage III, and IV are treated by 12 cycles of combination chemotherapy, MOPP (Mustin, Oncovin, Procarbazine & Prednisone).	**1. Radiotherapy and combination chemotherapy :** according to the stage of the disease. **2. Surgery : Indication :** (a) Gastric or intestinal lymphoma. (b) Staging laparotomy.
• **Prognosis :** An average 80% 5 years survival is obtained with proper treatment	

III- Burkitt's lymphoma



Etiology : Unknown may be related to infection with **Ebstein Barr (EB) virus** (Malaria may have a role).

Clinical Features :

- Common below the age of 12 years, in western Africa.
- The usual presentation is **painless progressively enlarging jaw swelling**
- It may also affect the kidneys, retroperitoneal tissues, ovaries, long bones & CNS.

Treatment : Chemotherapy. *Further cycles are needed to prevent recurrence*

N.B: pain in Hodgkin lymphoma is late and may occur with drinking alcohol.

N.B.

Hodgkin's disease

1. The neck is the preferred site for LN biopsy.
2. In LN biopsy, we should take the whole LN without rough manipulation to preserve its architecture.
3. Splenectomy is important to spares the kidney and lung the hazards of radiation injury.
4. Radiotherapy is used as a supplement to control bulky cervical LN.
5. 5-year survival rate is 100% with proper treatment of stage 1A with lymphocytic predominance.

Non-Hodgkin's disease

1. It has higher incidence with AIDS.
2. It may be related to infection with HTLV-1.
3. B cell lymphoma is the commonest type.
4. LN enlargement is similar to that of Hodgkin's disease.
5. Staging laparotomy is rarely indicated.
6. Mycosis fungoides is a variant of NHL in which skin eruption is the 1st of the disease manifestation.
7. Surgery is followed by radiotherapy and chemotherapy.
8. Gastric lymphoma has a better prognosis than gastric adenocarcinoma.

SECONDARY DEPOSITS

- The primary growth may be in a hidden site and may be overlooked, as in the ear, hypopharynx, nasal sinuses, nasopharynx, bronchi, stomach and testis.
- The affected glands are stony hard, painless and mobile at first, then become fixed and painful and may ulcerate and fungate through the skin

Lymphedema

Definition : Chronic edema from chronic lymphatic obstruction.

Sites : Limbs, breast, scrotum or vulva. In the limbs it affects the subcutaneous tissues.

Causes :

[A] Congenital (1ry lymphedema) : Rare

1. Congenital aplasia, hypoplasia or varicosity of lymphatics.
2. It may be familial **(Milroy's disease).**
3. It may manifest at birth (lymphedema congenita), at puberty **(lymphedema precox),** or in adults **(lymphedema tarda).**

[B] Acquired (2ry lymphedema) : Common

1. Traumatic : (a) Circumferential skin loss.
(b) Extensive block dissection of inguinal or axillary nodes.

2. Parasitic : *Filariasis is the commonest cause in Egypt.*

3. Inflammatory : (a) Chronic (recurrent acute) lymphangitis.
(b) Chronic specific lymphangitis e.g. T.B.

4. Neoplastic : (a) Primary affection of lymph nodes, as lymphomas.
(b) Primary affection of lymphatics as lymphangiosarcoma
(c) Secondary affection of lymph nodes by metastases.

Pathology : The pathology passes into 4 stages :

1. Stage of soft pitting edema : Due to accumulation of large amount of protein rich fluids in tissues.

2. Stage of lymphorrhea : Due to rupture of lymphatic vesicles discharging their lymph.

3. Stage of fibrosis (non-pitting edema) : Extravasated fluid with its high protein content excites fibrosis of skin and subcutaneous tissues.

Lymph stasis predisposes to recurrent lymphangitis. Each attack obliterates more lymphatics, so that limb edema increases steadily.

4. Stage of Elephantiasis : The skin is roughened, puckered and non-pitting like elephant skin.

Pathological changes:

1. Swelling.

2. The skin changes : In primary lymphedema skin changes are negligible. In long-standing secondary lymphedema, there is thickening and hyperkeratosis of the skin. Lymphatic vesicles may appear in the skin. It is to be noted that although the skin is very much thickened, it is not liable to ulceration as the diffusion of nutrients is normal (compare with venous ulcers in postphlebitic limbs).

Complications :

1. Recurrent cellulitis and lymphangitis (the most frequent).
2. Lymphedema ulcers from ruptured and infected blebs.
3. Huge disabling heavy limb.
4. Lymphangiosarcoma (very rare).

Differential diagnosis :

Other causes of chronic diffuse limb swelling : postphlebitic limb, elephantiasis neurofibromatosis and congenital arterio-venous fistula (local gigantism).

Clinical Picture :

1- Type of patient : it differs according to history of the cause e.g. in case of filarial lymphedema: adult patient from an endemic area (e.g. Giza, Imbaba. Rasheed, Damietta, Sharkaya and Assyot) complaining of progressive leg swelling with exacerbations and partial remission.

2- During exacerbations : (Attacks of streptococcal lymphangitis) the limb shows red tender streaks traveling to draining L.N.s.

3- The swelling : is hard non-pitting with **a crease in front of the ankle(stemmer's sign):**this is because the skin over the ankle drains directly to deep lymphatics.

4- In late cases :

(a) The skin may show blebs, which may rupture → superficial ulcers oozing lymph (lymphorrhea).

(b) Skin is dark, thick with multiple deep furrows, thick callosities & warty over growths (Elephantiasis)

5- The inguinal L.N.s : may be enlarged, firm and tender (chronic septic lymphadenitis) or may form a soft lobulated mass (varicose L.N.s).

6- Other filarial manifestations : as lymphedema of scrotum, filarial funciulo-epididymitis, chylocele (rupture of lymphatics in tunica vaginalis), chylous ascitis and chylothorax.

Investigation :

1. Night blood film : between 10pm - 2am- may show microfilaria in active filariasis.

2. Blood picture : Shows esinophilia.

3. Intra dermal tests.

4. Inguinal L.N. Biopsy : May show dead filaria worms, calcification or malignant infiltration in case of malignant deposits.

5. Lymphangiography.

6. Lymphoscintigraphy : by using technetium 99^{m}.

Treatment :

[A] Conservative treatment : Indication : Mild and moderate cases.

Method : 1. Limb hygiene, elevation, elastic stockings and diuretics.
2. Long acting penicillin 1.200.000 units every 3 weeks.
3. Active filarial infection is treated by diethyl carbamazine
- Dose : 1mg/kg increased over 3 days to 6mg/kg/day for 21 days.

[B] Surgical Treatment :

I- Physiological Operations :

1. Enteromesenteric bridge operation : A segment of the ileum with its mesentery is separated and is brought under the inguinal ligament. The mucosa of the bowel is removed and the opened bowel is sutured over the cut lymph nodes.

2. Omental flap operation : A segment of the greater omentum is mobilized with intact blood supply and is transferred into the lower limb to increase the lymphatic drainage.

3. Microlymphatico-venous anastomosis : The dilated obstructed lymphatic trunks are anastomosed to nearby veins.

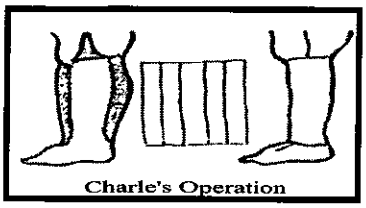
4. Microlymphatic transfer operation : healthy lymphatic trunks are harvested from the normal limb & anastomosed to bypass the obstructed lymphatics.

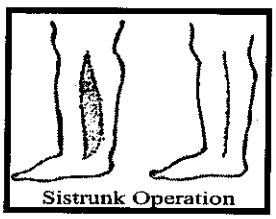
5. Lympho-venous anastomosis : A lymph node is bisected and anastomosed to a nearby vein.

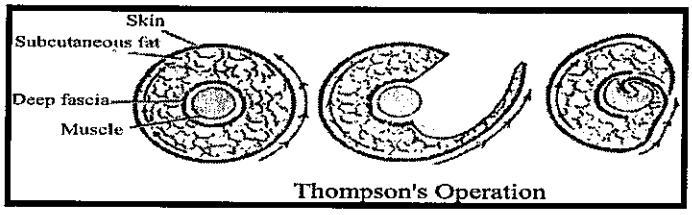
II- Excisional operations :

1. Charles operation : Excision of the skin & SC tissues + Covering the limb by split skin graft.

2. Sistrunk operation : Excision of an ellipse of skin & SC tissues + Closure of the defect. This decreases the bulk of the limb at one side. This can be repeated on the other side at later date.







III- Physiological and excisional operations :

- **Thompson operation (Swiss roll operation) :** Thompson's operation consists essentially of excision of subcutaneous tissue and then implantation of a shaved flap of skin between the muscles near the deep lymphatic vessels so that the physiological drainage may be improved.

Filarial Lymphedema

This is the commonest cause of lymphedema in tropics. It occurs where culex pipiens mosquito (intermediate host) exists. Very heavy and repeated infestation by mosquito bites is essential for pathology to start.

Cause : Wuchereria Bancrofti.

Life cycle : In man **(definitive host)** the mature worms live in the lymphatics and lymph nodes. The microfilaria are laid by the female, and then circulate in the peripheral blood at night. They have a nocturnal periodicity (between 10 pm. - 2 am. with the peak round midnight). The microfilaria are sucked by the mosquito culex pipiens **(intermediate host).** In the body of mosquito, the microfilaria passes to stage of embryofilaria **(infective stage)** where it is injected into the body of patient by bite of mosquito. The embryofilaria maturate to adult worms.

Pathology : Adult worms live in the cortex of lymph nodes obstructing the outflow of lymph. This produces cystic enlargement of lymph node, which is given the name of varicose gland.

- **Role of filaria worms is Just to start attacks of streptococcal lymphangitis by causing lymph stasis. Big number of worms is essential. Once lymphangitis starts, the disease continues automatically like any lymphedema, whether worms are living or dead.**

Peripheral Nerves

Peripheral Nerves

Nerve Injuries

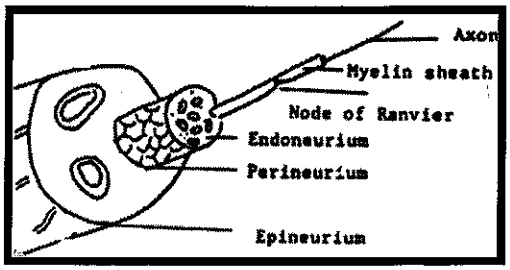
Causes of nerve injury :

[A] Open injuries : As a result of stabs, bullets and lacerated wounds or deep burns.

[B] Closed injuries : As in fractures and dislocations.

- It may be due to :

1. Compression from outside as by tourniquet, plaster of paris, edge of operating table (as in radial nerve injury).
2. Traction Injuries as birth injury.
3. Ischemic injuries.
4. Contusion by a direct blow.
5. Chemical irritation as by accidental injection of irritant drugs in a nerve.



Pathology :

1. Neuropraxia : (concussion of nerves). There is momentary loss of conduction through the nerve without any organic lesion (nerve fibers are intact). No degeneration. Recovery is complete and quick.

2. Axontmesis : This injury involves the axon without loss of continuity of nerve sheath. Wallerian degeneration occurs. There is complete motor and sensory paralysis. Recovery is usually complete as the sheaths are intact.

3. Neurotmesis : Partial or complete division of a nerve. Degeneration occurs with the formation of terminal or lateral neuroma. Spontaneous recovery is impossible, recovery only after treatment and usually incomplete (due to maldistribution of fibers).

Effects of total division of a mixed nerve :

[1] Motor effects :

1. Loss of voluntary contraction.
2. Muscle atrophy and wasting.
3. Deformity due to stretching of the paralyzed muscle by the unparalyzed opponent (e.g. Claw hand).
4. Loss of reflexes.

[2] Sensory changes :

1. Anesthesia in the area supplied by the nerve (usually less than the anatomical distribution due to overlap of dermatomes).
2. Pain referred along the distribution of the injured nerve.

[3] Vasomotor changes :

1. Primary vasodilatation due to paralysis of vasomotor fibers.
2. Later there is vasoconstriction from loss of the afferent part of vasomotor reflex.

[4] Pseudomotor : Loss of sweating = Anhydrosis.

[5] Trophic changes :

1. **Skin** becomes thin and shiny with loss of subcutaneous fat.
2. **Nails** become distorted and brittle.
3. **Bone** becomes decalcified.

[6] Changes in the nerve :

1. Retraction of divided ends.

2. Wallerian degeneration occurs below the injury, and above as far as the first **Node of Ranvier.** Degeneration starts 4 days after injury.

3. Neuroma formation : It is formed of the growing axons and fibrous tissue reaction, which give a swelling wrongly termed neuroma. It may be in the center of the nerve (central), on one side (lateral) or at the end of the nerve (terminal neuroma).

Diagnosis of nerve injury :

[A] Clinical picture : History of trauma and its type. Examination for the effects (see before).

[B] Tests :

1. Sweating test : to map out the area of anhydrosis using **Quinizarine powder.**

2. X-ray : for detection of foreign bodies and fractures.

3. Nerve conduction velocity (NCV) : Neuropraxia does not interfere with NCV. However, after complete transection of a nerve, NCV progressively decreases. With regeneration NCV will gradually increase. It is useful in diagnosis of compression neuropathy and in detecting regenerating axons.

4. Electromyography : Two to four weeks after complete nerve injury, the denervated muscle shows spontaneous fibrillations **(fibrillation pattern).** These fibrillations remain as long as the muscle is still viable, but if complete fibrosis occurs, no electrical activity is detected and the muscle is no longer suitable for reinnervation.

Treatment :

[A] Conservative treatment :

Indications :

- In closed injuries : in neuropraxia and axontmesis.
- During nerve repair until reinnervation occurs.

This consists of :

1. Splintage : To prevent over stretching of the paralyzed muscles.

2. Galvanic stimulation to prevent atrophy and wasting of paralyzed muscles.

3. Physiotherapy.

[B] Operative treatment :

Indications :

- In open injuries.
- In closed injuries, which fail to recover within the expected time of recovery.

This consists of :

[1] Nerve repair :

(a) In clean incised wounds :

- Immediate primary suture is the ideal treatment as it gives the best results.

(b) In contaminated or lacerated wounds :

- Primary nerve suture should not be attempted.
- **Secondary nerve suture is performed after complete healing of the skin 2-3 weeks as :**

1. Dissection opens new planes for infection.
2. The nerve sheath is too thin to hold sutures, after 2-3 weeks the nerve sheath is toughened.

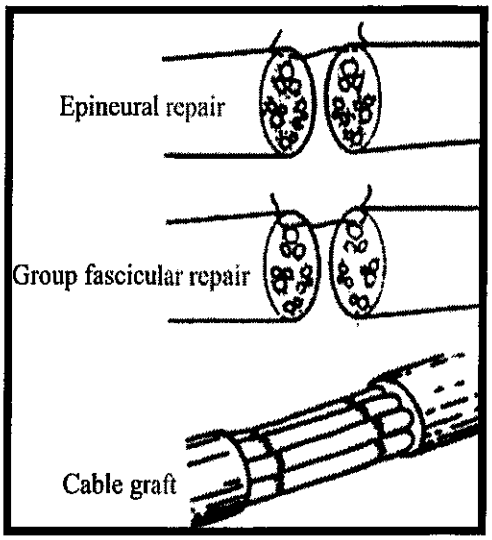
- The ends of the divided nerve are loosely approximated with single stitch of silk.

Technique :

1. If the nerve is constricted between fragments or in scar tissue, it is mobilized to relieve any pressure or constriction **(neurolysis).**

2. If the nerve is obviously completely divided or the seat of a firm bulbous neuroma, nerve suture is indicated :

- The nerve trunk is clearly divided immediately above and below the damaged area. Accurate apposition is obtained by **"Interrupted 6/0 prolene sutures".** The sutures pick up the nerve sheath only (epineurium). This type of repair is called **"Epineural repair".** A more accurate repair called **"Interfascicular repair"** is performed using the **"Operating microscope"** where the fasciculi of the proximal and distal segments are very accurately opposed by passing the sutures through the perineurium.



Post-operative care :

1. Plaster fixation is maintained for 3 weeks.
2. Care of the denervated structures continues until recovery of function.
3. Rehabilitation.

Results : The prospects of success depend on several factors :

1. The injured nerve. The radial nerve is the most satisfactory.
2. Level of suture.
3. Time interval between injury and suture.
4. Extent of injury.
5. Age and general condition of the patient. The results of nerve suture are particularly good in children and adolescents.

Only if the approximation of the two ends is difficult, one of the following methods is done :

1. Flexion of the limb.
2. Mobilization of the nerve proximally and distally.
3. Stripping of unimportant anchoring branches.
4. Transposition of the nerve to shorten its course.
5. Bone shortening.
6. Nerve grafting either by using a less important nerve or multiple strands of cutaneous nerve are used as a bundle (Cable graft).

[2] Orthopedic treatment :

- Is used when one fails to make the nerve to recover. **It consists of :**

1. Arthrodesis : Fixation of the joint.
2. Tendon transplantation.

Brachial Plexus injury

Etiology :

1. Open injuries.
2. Traction injuries.
3. Pressure injuries e.g. Fractures of the clavicle.

Clinical Features : Three types of lesions are present :

[1] Whole plexus type : Rare

- The whole arm is paralyzed, flaccid & anesthetic by the patient's side.
- Sensation is present only on the medial side of the arm (intercosto-brachial nerve).
- There may be sympathetic paralysis of the eye and face producing ptosis, myosis, enophthalmos and anhydrosis of one side of the face **(Horner's syndrome).**

[2] Upper arm type (Erb-Duchenne paralysis) :

The 5th & sometimes 6th cervical nerves are involved :

- The limb hangs by the side with the forearm extended and pronated in the so-called **"policeman tip position".**
- The arm can't be abducted or rotated, the elbow cannot be flexed & the forearm can't be supinated.
- The movements of the hand and wrist are not impaired.
- Sensory changes are absent if the fifth nerve only is involved, but if the sixth nerve also suffers, an area of anesthesia is present over the outer side of the arm.

[3] Lower arm type (Klumpke's paralysis):

The 1st thoracic & sometimes the 8th cervical nerves are affected :

- Paralysis of the intrinsic muscles of the hand (1st thoracic).
- **Claw hand** with wasting of small muscles of the hand.
- Anesthesia along the inner side of the forearm and inner 3 & 1/2 fingers.
- **Horner's syndrome** may occur.

Treatment :

- Conservative treatment.
- Exploration of the plexus is indicated in lesions which fail to show any evidence of recovery within 2 months.

Circumflex nerve injury

Causes : Gunshot wounds, direct blow, fractures of surgical neck of humerus or crutch palsy.

Clinically : Deltoid and teres minor are paralyzed leading to **loss of abduction of shoulder (between 15- 90),** and shoulder is flat (due to wasting of deltoid).

Radial nerve injury

Etiology :

1. Injury in the axilla : may result from pressure by crutches or may follow fractures and dislocations of the upper end of the humerus or attempts at their reduction.

2. Injury in the spiral groove may be due to :

(a) Fractures of the shaft of the humerus.
(b) The edge of a chair in Saturday-night paralysis.
(c) Prolonged application of a tourniquet.
(d) Operation in which the outstretched arm has rested on the edge of the table.
(e) Intramuscular injections of irritant drugs.

3. Posterior interosseus nerve : may be injured in fracture head of radius.

Clinical picture :

[A] Injury in the axilla :

1. Motor paralysis :

(a) Paralysis of triceps (uncommon) leading to flexion of elbow (extended only by weight of forearm).

(b) Paralysis of supinator and brachioradialis. As a result the forearm is pronated (biceps can act as supinator on flexion of elbow).

(c) Paralysis of extensors of wrist leading to **wrist drop** and paralysis of extensors of the fingers leading to **finger drop** with flexion at metacarpophalangeal joint. As the lumbricals and interossei are not paralyzed, extension of the terminal 2 phalanges can be done.

[B] Injury in the spiral groove :

1. Motor paralysis : is similar to those following injury in the axilla except that the triceps and anconeus muscles escape.

2. Sensory loss : Anesthesia is limited to a patch on dorsum of the hand and thumb.

[C] Injury in the elbow :

1. Motor paralysis : Posterior interosseus nerve injury. The elbow can be extended and supination is free. Finger drop is present; the wrist can be extended by Extensor carpi radialis longus.

2. Sensory loss : Small area on radial side of the back of hand and thumb.

N.B: The grasp is weak since the flexion cannot be tightened by extending the wrist joint.

Treatment :

1. Conservative : Keep the wrist and fingers in extension on a **"cockup"** splint.
Massage, electric stimulation.

2. Surgical : If no improvement occurs in 1.5 months. Secondary nerve suture is done. If there is loss of nerve substance, the radial nerve is transposed to the front of humerus. **(Good results are obtained, as the nerve is almost purely motor).**

3. Tendon transplantation : If nerve suture fails. Grafting the tendon of flexor carpi ulnaris to extensors of the fingers and flexor carpi radialis to extensors of thumb.

Median nerve injury

Causes : Cut wounds above the wrist, fractures of lower end of radius and dislocation of lunate bone.

Clinical picture :

[A] Injury above the wrist :

1. Motor paralysis : Paralysis of all muscles of thumb except adductor pollicis (which is supplied by ulnar nerve). The thenar eminence is flattened; the thumb is adducted like the thumb of apes **(Ape hand).** The thumb can't be abducted or opposed.

2. Sensory loss : Over the lateral 3 & 1/2 fingers and lateral 3/5 of the palm.

3. Trophic changes : Loss of the pulp fat leading to tapering of the fingers.

[B] Injury in the forearm : Same like above and in addition there is :

1. Motor paralysis : Of the 2 pronators leading to loss of pronation.

2. Loss of flexion of the distal phalanx of index finger (paralysis of profundus), loss of flexion of middle phalanx (paralysis of superficialis) and lost flexion at metacarpophalangeal joint (paralysis of 1st lumbrical muscle). Thus in **clasping test** the index is permanently extended and tapering. **This is called pointed (thin) pointing (extended) finger.**

3. Loss of flexion of the terminal phalanx of thumb (due to paralysis of flexor pollicis longus).

4. Causalgia : Severe burning pain along the distribution of median nerve injury. This pain is relieved by sympathectomy.

Prognosis :

In median nerve injury is poor as it is a mixed nerve and supplies muscles, which control the movements.

Ulnar nerve injury

The nerve is either injured at the elbow or wrist

[A] Injury at the elbow : Causes :

1. Fractures of the medial epicondyle. 2. Excision of the elbow joint. 3. Wounds at the elbow.
4. Cubitus valgus due to old injury of the humerus and increase of the carrying angle. The ulnar nerve is unduly stretched and continous friction leads to interstitial neuritis.

Clinical picture :

1. Motor paralysis :

(a) The flexor carpi ulnaris and medial 1/2 of profundus are paralyzed. This results in hyperextension at the metacarpophalangeal joint (as the flexors are weak).

(b) Paralysis of all short muscles of the hand except 1st and 2nd lumbricals, flexor, opponence and abductor of the thumb.

Froment's test : If the patient is asked to hold a piece of paper between his thumb and fingers, the terminal phalanx of thumb is flexed, as the patient is unable to adduct the thumb. Paralysis of interossei leading to wasting and inability to adduct fingers (tested by holding a paper between fingers).

2. Sensory loss : Over the medial 1 1/2 fingers and medial 1/3 of hand infront and behind.

3. Trophic changes : Marked.

[B] Injury at the wrist :

Causes : are the same causes as those of injury of median nerve at wrist.

Clinical picture :

1. Motor : Paralysis and wasting of small muscles of the hand.
2. Sensory loss : On the anterior aspect of the inner 1 1/2 finger.
3. Trophic changes : correspond to area of sensory loss.

Treatment : General principles are applied but prognosis is bad.

N.B.: A cut wound at the front of the wrist may injure the ulnar and median nerves, one or more of the flexor tendons in addition to the blood vessels.

Injuries of nerves of the lower limbs

Items	Sciatic nerve	Lateral popliteal nerve	Medial popliteal nerve
Causes of injury	1. Wounds, fractures, dislocation of hip or IM injection of drugs. 2. It may be paralyzed while it is still in the pelvis by tumors or aneurysms.	1. Fracture neck fibula (or its excision). 2. Pressure from plasters or splints. 3. Wounds.	
Motor	1. Paralysis of flexors of the knee (but some degree of flexion can be done by sartorius and gracilis). 2. Complete paralysis below knee.	Complete paralysis of the extensors and peroneal muscles leading to Talipes equino-varus deformity.	Paralysis of muscles of calf and sole leading to Talipes calcaneo-valgus and clawing of toes.
Sensory	Complete sensory loss below knee with the exception of skin supplied by Saphenous nerve (inner side of leg, inner border of foot and big toe).	Over the dorsum of the foot and part of the front and outer side of leg.	Sensory loss over the sole.
Sympath-etic	Changes on the sole and toes & Causalgia in partial nerve injury.		Severe trophic changes & Causalgia in partial nerve injury.
Treatment	General principles are applied.		
Prognosis		Good in early nerve suture (Nerve is mostly motor).	

Neurofibromatosis

Definition :

- It is a congenital disease affecting nerves and the resulting tumors arise from fibrous tissue of the neurilemmal sheath

Types of neurofibromatosis :

1. Generalized type : (Von Recklinghausen's disease)

- It may affect any nerve trunk **(cranial or spinal).** Multiple tumors all over the body occur.
- Each tumor is painless but tender **(due to pressure on, and not infiltration of, the affected nerve).**
- The swelling is fusiform in shape; firm in consistency, mobile across but not a long the nerve trunk.
- No interference with nerve function i.e. no spontaneous pain, anesthesia or paralysis of motor nerves. **(Any interference with nerve function means sarcomatous transformation).**
- Brownish pigmentation of the skin is associated **"Café au lait"** patches
- **Treatment :** Removal only of tumors causing pain or pressure effect.

2. Solitary neurofibroma :

- Affecting one nerve trunk **(cranial or spinal).**
- The swelling is firm, tender, moves across but not a long the line of the nerve.
- It may turn malignant and must be removed.

3. Acoustic neuroma :

- Solitary neurofibroma arising from acoustic nerve leading to deafness, cerebellar symptoms and increased intracranial tension.
- It may be solitary or as a part of the generalized disease.

4. Molluscum fibrosum :

- It is a neurofibroma arising from the termination of cutaneous nerves.
- It affects any part of the skin except the palms and soles.

5. Plexiform neuroma :

- It affects subcutaneous nerve plexuses.
- The skin is redundant and the thickened nerves and neurofibromata are felt as multiple firm nodulated cords.

6. Elephantiasis neuromatosa :

- Marked thickening of skin and subcutaneous tissue of a part of the body (commonly a limb).

7. Neurofibrosarcoma :

- May arise de-novo or on top of a neurofibroma. It is a fibrosarcoma arising from fibrous tissue of neurilemmal sheath.
- The tumor grows rapidly. Local infiltration occurs early and distant metastasis may occur.
- There is spontaneous pain, interference with nerve function (anesthesia and paralysis).
- **Treatment :** Of neurosarcoma is wide excision or amputation

Thyroid Diseases

Thyroid Diseases

Thyroglossal cyst & fistula

Thyroglossal cyst

Etiology: Congenital, due to persisting part of thyroglossal duct.

Incidence:

- **Age of onset:** Although congenital it is not present since birth but appears in childhood (It needs time to enlarge).

Pathology: **Structure of Thyroglossal cyst:**

- The cyst is lined with columnar epithelium.
- Its wall is rich in lymphatic tissue communicating with cervical nodes making the cyst liable to infection.
- The cyst contains clear mucoid fluid rich in cholesterol crystals.

Clinical Picture:

1. Size : Small (2-3 cm diameter).

2. Site : Commonly subhyoid, less commonly on thyroid cartilage and rarely suprahyoid. It lies in midline, but if on thyroid cartilage it is pushed by the central prominence of larynx to one side, usually the left.

3. Mobility :

- Being attached to foramen caecum, it moves up with protrusion of tongue (diagnostic).
- In addition it moves up with swallowing as goitre, being inside pretracheal fascia.

N.B.:

- **It is attached to hyoid bone by a fibrous cord.**

D.D.: From midline neck swellings (cystic and solid), but mainly from cystadenoma of thyroid isthmus.

Complications: **Liable to repeated infection.**

N.B.:

- Thyroglossal cyst & subhyoid bursitis moves with deglutition & protrusion of tongue.

Thyroglossal fistula

Never congenital, but results from :

1. Incomplete removal of thyroglossal cysts.
2. Rupture or incision of the inflamed cyst.

Clinically :

1. History of pre-existing cyst.
2. The fistula opens in midline, low in neck.
3. The opening shows a characteristic semilunar fold of skin due to retraction of the fistula as the neck elongates.
4. It discharges mucus and, if infected, pus.
5. The track of the fistula can be felt as cord attached to body of hyoid bone and moves up with protrusion of tongue and also with swallowing.

D.D.: From fistulae and sinuses in neck.

Structure :
The thyroglossal fistula is lined by columnar epithelium and discharges mucus. Its wall is rich in lymphoid tissue and is liable to recurrent inflammation.

Treatment :
The same for cyst of fistula. The idea is to remove all remnants of thyroglossal duct up to foramen caecum **(Sistrunk's operation),** the fistula or cyst is dissected and the track is followed up to hyoid bone. The body of hyoid bone is excised with the track, being adherent to it. Finally, the track is followed up to foramen caecum in tongue.

N.B. Recurrence after operation occurs as a thyroglossal fistula. This is due to incomplete removal of thyroglossal duct.

Lingual thyroid

Pathology :
Failure of descent of the thyroid can lead to an intralingual development of the gland.

Clinical picture :
Patients may present with a tongue swelling with obstructive manifestations i.e. dysphagia, difficult breathing or change of quality of voice.

Investigations :
Thyroid scans: show absence of thyroid tissue in the normal side in the neck.

Treatment :
1. Full replacement with L-thyroxine to induce reduction in size.
2. Surgical excision
3. Radioactive-iodine ablation

Mechanism of formation of thyroid hormones

T Iodine **T**rapping.
O **O**xidation by peroxidase enzyme (inorganic → organic).
B **B**inding to tyrosine → Mono & di-iodotyrosine (MIT, DIT)
C **C**oupling of MIT & DIT → T3, T4
S **S** torage as thyroglobulin.
R **R**elease.

N.B :
- **Perchlorate** → prevents T
- **Carbimazol & Thyouracil** → depress B
- **Lugol's iodine** → depress R
- **T.S.H** → +++T, O, B, C, S, R & produces hyperplasia of the gland

N.B. : The Pituitary–Thyroid Axis :
- T.S.H secretion is controlled by blood level of thyroxin. A low blood level of thyroxin stimulates the release of T.R.H (thyrotropin releasing hormone), which stimulates the anterior pituitary to produce T.S.H. The later increases the size of the thyroid gland, its vascularity and its production of thyroid hormones.
- A high level of thyroxin on the other hand inhibits TRH & T.S.H and reduces the release of thyroxin.

Goitre

What is goiter?

In Latin-gutter=the throat
It is any generalized enlargement of thyroid gland.

Types of Goitre:

1. Simple:
(a) Physiological.
(b) Colloid.
(c) Nodular.

2. Toxic:
(a) Primary.
(b) Secondary.
(c) Toxic nodule.

3. Neoplastic: Thyroid carcinoma (benign tumors are rare).

5. Autoimmune goitre as Hashimoto's thyroiditis and collagen diseases as Riedel's thyroiditis
6. Hypothyroid goitre.

Simple Goitre

Definition :

Non-toxic, non-neoplastic, non-inflammatory thyroid enlargement. Essentially the patient is euthyroid.

Incidence :

Sex : Commoner in females.
Age : Usually starts at a young age especially in endemic areas.
Residence : Certain areas are endemic (oases), but sporadic cases appear anywhere.

Cause :

Simple goitre is due to persistent stimulation of thyroid gland by high TSH level. Increased TSH secretion is due to decreased production of thyroid hormones from:

[A] Iodine deficiency :

- This may be absolute or relative.
- The normal daily requirement of iodine is 100-125 µg.

1. Absolute deficiency : Simple goitre is endemic in regions with low iodide content of the water.
2. Relative deficiency : Occurs during periods of stress e.g. females during the menarche, pregnancy and lactation.

[B] Enzymatic deficiency :

- Responsible for many cases of sporadic goitre.
- Positive family history is common.
- Examples :

1. Pendred's syndrome : It is due to deficiency of peroxidase enzyme. There are goitre, deafness and mutism.
2. Deficiency of dehalogenase enzyme

[C] Goitrogens : e.g.: Thiocyanates in cabbages, drugs as para-amino salicylic acid and antithyroid drugs. Large doses of iodides inhibit iodine binding, thus resulting in iodide goitre.

Pathogenesis (How does simple goitre develop ?)

Stages of goitre formation :

[1] Deficient synthesis of thyroid hormone leads to persistent TSH elevation that produces diffuse homogenous hyperplasia. All follicles are active **(Diffuse hyperplastic goitre).**

- This stage is reversible, i.e. If the iodine requirements are satisfied, the follicles undergo involution.

[2] Sometimes, when the iodine requirements are satisfied the follicles undergo hyperinvolution and become large in size, lined by flattened epithelium and full of colloid **(Simple colloid goiter).**

[3] Repeated fluctuations in the level of TSH due to repeated stresses which produce :

(a) Repetition of this process of hyperplasia and involution.

(b) Heterogenecity of the gland, with areas of active follicles and others with inactive follicles.

(c) Nodular goiter : most nodules being inactive, the active follicles being present in the internodular tissue.

Immunoglobulins is one of the factors (other than TSH), which play a role in etiology of nodular goitre.

1. Physiological Goitre (Diffuse Hyperplastic Goitre)

Incidence : Usually in female at puberty (10-20y) during pregnancy and lactation.

Pathogenesis : See before.

Gross Features : Regular, moderate, smooth, soft enlargement of the whole gland cut section is fleshy brown.

Histology : Diffuse hyperplasia. Acini are increased in number, lined with columnar epithelium and contain little colloid.

Clinical picture : The only symptom is fullness of lower part of the front of neck **(Venus neck).**

Signs : (Thyroid swelling)

1. Regular enlargement of the whole gland, which is butterfly shaped.
2. Moderate size.
3. Smooth surface.
4. Soft consistency.

2. Colloid Goitre

Is an intermediate stage between physiological and nodular goitre.

Gross features : The gland is regularly or irregularly enlarged, soft and smooth. Cut section shows wide spaces filled with colloid.

Histology : Hyperinvolution. Acini are hugely distended with colloid and lined with flat epithelium. Adjacent acini may rupture and fuse together forming large cysts filled with colloid.

Clinical Picture :

The gland is enlarged with irregular surface and soft consistency.

3. Simple Nodular Goitre

Clinically there are 2 types :

Diffuse simple nodular goitre = More than one nodule is felt.
Solitary simple nodular goitre = One nodule is felt.

Incidence :

Frequency : It is the commonest type of goiter. **Locality :** sporadic or endemic.
Age : 30-40 (younger if endemic). **Sex :** Commoner in females.

Pathogenesis : See before.

Gross features :

- Usually multiple nodules are grossly present.
- A dominant nodule may be present but microscopic changes are present throughout the gland.
- Cystic degeneration, hemorrhage and calcification are common.

Histology :

- Dense fibrous stroma around nodules.
- Nodules contain hyperinvoluted acini (colloid nodules which are bulky & gelatinous), distended with colloid and lined with flat epithelium.

N.B.: Most nodules are inactive. Active acini exist only in internodular tissue.

Clinical picture :

Symptoms : There are 2 groups of symptoms :

1. Disfiguring swelling : In lower part of neck of insidious onset, long duration & gradual enlargement.

2. Pressure symptoms (by large goitre) :

[A] Dyspnea from pressure on trachea :

It increases by sleeping on high or low pillow, so that it is called postural dyspnea. **It results from :**

1. Lateral kinking of trachea in unilateral goitre,
2. Compression from both sides in bilateral huge goitre (scabbard trachea)
3. Anteroposterior compression in retrosternal goitre.
4. Prolonged pressure on trachea produces softening and easy collapse (tracheomalacia).

[B] Dysphagia from pressure on esophagus : only retrosternal goitre.

Signs : The swelling shows the following characters :

1. **Butterfly shape** (Diffuse enlargement) or **Localized lump** (adenoma).
2. **In lower part of front of neck** (Muscular triangle).
3. **Moves up with deglutition** (being enclosed in pretracheal fascia).

N.B.: These 3 signs prove that the swelling is goiter

> Through pretracheal fascia, the thyroid gland is suspended to thyroid cartilage & hyoid bone, which moves with deglutition. Ligament of Berry is a thickening of posterior part of pretracheal fascia that fixes the back of each lobe to the cricoid cartilage.

Swellings move with deglutition	Thyroid swellings don't move with deglutition
Thyroglossal cyst, cervical ectopic thyroid, prelaryngeal & pretracheal L.N.s, subhyoid bursitis, parathyroid swelling.	Fixed by malignancy, Reidel's thyroiditis, scarring of previous operation, huge goiter.

4. **Size :** Variable.
5. **Surface :** Nodular.
6. **Consistency :** Firm or tense cyst.
   - If hard = calcification, malignancy or Reidl's thyroiditis.
   - If cystic = cystic degeneration, hemorrhage or suppuration.
7. **Border :** Well-defined (in retrosternal goitre: lower border can not be felt).

8. Tenderness : Not except if infection occurs in a nodule.
9. No thrill or murmur over the gland (only in toxic goitre).
10. Transmitted pulsations may exist (expansile in toxic goitre).
11. Percussion over manubrium sterni = dullness in retrosternal goitre.
12. Relation of the swelling to surrounding structures :
- **Absent carotid pulse denotes malignant infiltration = Berry's sign.**
- **Fixity of goitre to skin, sternomastoid or trachea denotes thyroiditis, malignancy or postoperative adhesions.**

Investigations :

1. Routine lab & ECG.
2. Indirect Laryngoscopy.
3. Plain X-ray : may show calcification and tracheal deviation or compression.
4. Ultrasound : to detect cystic from solid lesions and can it can detect retresternal goiter
5. Thyroid function tests : to exclude, hyper or hypothyroidism(subclinical case of toxic goiter).
6. Thyroid antibodies estimation : to exclude autoimmune (Hashimoto's) thyroiditis : not routine.
7. Biopsy (FNAC) : not routine.

Complications of simple nodular goiter :

1. Malignancy : Recent rapid enlargement + hard consistency (3%). Usually follicular type.
2. Toxicity : producing secondary toxic goitre (30%).
3. Tracheal obstruction by compression : (see c/p).
4. Infection in a nodule : Rapid enlargement, pain, warmth & tenderness.
5. Cyst formation (cystadenoma) from :
(a) Degenerated nodule.
(b) Rupture and fusion of adjacent colloid acini.
6. Hemorrhage in a nodule or cystadenoma :
Produces sudden enlargement of preexisting goitre. Severe dyspnea results if impaction occurs at thoracic outlet **and urgent treatment is essential :**
(a) Cold applications. (b) Aspiration.
(c) Incision in skin and deep fascia allowing the gland to bulge freely in the wound releasing the trachea until urgent thyroidectomy is done.
7. Calcification in a nodule : hard, simulating malignancy.
8. Retrosternal extension : occurs usually in men due to stronger pretracheal muscles and shorter neck.

Treatment of simple goiter :

1. Prevention: The use of iodized table salt (potassium iodide 1:10,000)

2. Treatment of diffuse hyperplastic goitre:

- Usually reversible by the use of L-thyroxin, usually 0.2 mg/d for several months to be tapered to 0.1 mg/day for several years.
- The patient is reassured and is advised for regular follow-up.

3. Treatment of simple nodular goiter:

Surgery is indicated for :
1. Evidence of compression manifestations mainly upon the trachea.
2. Cosmetic reasons.
- Both lobes affected = Partial thyroidectomy (subtotal).
- Only one lobe affected = Hemi-thyroidectomy.

N.B.:
1. Removed gland is sent for histological examination (biopsy).

N.B: Histological surprise:
Accidentally discovered malignancy from histological examination of removed benign thyroid swelling.

2. After operation 0.1mg L-thyroxin is given daily for life to avoid recurrence by inhibiting TSH stimulation.
3. Surgery should not be advised below the age of 25 unless very much indicated as it may be followed by recurrence.

Retrosternal Goitre

Definition :

- Goitre in superior mediastinum.
- It is an anatomical, not pathological diagnosis.

Pathology :

- Retrosternal goitres usually arise in a normally placed thyroid gland. The sternohyoid and sternothyroid muscles prevent forward expansion, and assisted by gravity and the negative intrathoracic pressure, they direct the swelling into the mediastinum.
- In most cases, the intrathoracic swelling occupies the superior mediastinum, but occasionally it lies in the posterior mediastinum behind the trachea, and sometimes behind the esophagus.

Retrosternal goitre is classified into 3 types :

1. Plunging goitres : which rise with deglutition and descend again through the thoracic inlet.
2. Mediastinal goitres : which lie wholly in the chest, but are connected with the thyroid by a relatively narrow band of tissue and derive their blood supply from the thyroid vessels.
3. Intrathoracic goitres : which lie wholly in the chest, completely separate from the gland. They probably arise from congenitally misplaced thyroid tissue, deriving independent blood supply from mediastinal vessels.

Clinical features :

- An intrathoracic goiter is more common in men particularly short-necked individuals.
- It often remains symptomless for years.

1. **Dyspnea** from compression of the trachea, it gets worse at night and aggravated with lying down or flexion of the neck. Some patients are diagnosed as asthamtics.
2. **Cyanosis and edema** of the face and neck.
3. **Dilatation of superficial veins of the upper part of the chest** due to obstruction of the innominate veins **(D.D.** congestive heart failure**).**
4. **Dysphagia** may be present.
5. **Palpation of the neck** may reveal an enlarged thyroid gland (the lower edge cannot be felt).
6. **Percussion of the sternum** may reveal retrosternal dullness.
7. **Symptoms and signs of toxicity or malignancy** may be present.

Special tests : (Pemberton's sign)

Ask the patient to raise up both arms or to tilt her/his head to one side strongly & keep this position for a while → Congestion of face & distress occurs due to obstruction of great veins & trachea at thoracic inlet.

Investigations :

1. Chest X-ray shows a soft tissue shadow in superior mediastinum.
2. Sonar.
3. Thyroid scan.
4. C.T. Scan of thorax will relieve the exact level of the retrosternal extension and its anatomic relations.

Treatment :

Surgery is always essential. Cervical part of goitre is dealt with as usual and its vessels ligated. This step avoids the risk of hemorrhage, as retrosternal goitre takes its blood supply from neck.

Retrosternal goitre is dealt with by :

1. Gentle pulling to deliver it in neck.
2. Piecemeal removal from within the capsule to collapse the goitre and then pull it up in neck.
3. Only if retrosternal goitre is malignant or intrathoracic, sternal split is needed (median sternotomy).

N.B.: Preparation of toxic cases is only by Inderal, as antithyroid drugs are (goiterogenic).

Toxic Goitre (Thyrotoxicosis)

The term thyrotoxicosis is better used, as not all manifestations are a direct effect of elevated thyroid hormones, e.g. True Exophthalmos and pretibial myxedema.

Common types :

- Diffuse toxic goitre (Primary toxic goitre = Grave's disease) 75%.
- Toxic nodular goitre (Secondary toxic goitre = Plummer's disease) 15%.
- Toxic nodule 5%.

Rare causes of hyperthyroidism :

1. Thyrotoxicosis factitia : due to excess intake of L-thyroxin (> 0.2 - 0.3 mg per day).
2. Jod-Basedow thyrotoxicosis : Occurs in cases of physiological goitre upon the administration of large doses of iodine (iodine induced toxic goitre). Temporary
3. Subacute thyroiditis : De Quervain's diseases.
4. Hashitoxicosis : 5% of patients with Hashimoto's disease may have hyperthyroidism in the early stages of the disease.
5. Functioning thyroid carcinoma.
6. Neonatal thyrotoxicosis : may occur in newborns of thyrotoxic mothers due to transmission of thyroid antibodies across the placenta. Subsides in 3-4 weeks
7. TSH secreting adenomas of pituitary gland
N.B:T4 pseudothyrotoxicosis: there is increased T4 and decreased T3 secondary to failure of peripheral conversion of T4 to T3.

Primary toxic Goitre

There is diffuse enlargement of the whole thyroid gland accompanied by the clinical picture of thyrotoxicosis.

Incidence :

- Females : Males = 8:1.
- Usually 20 - 40 years.
- Usually associated with unilateral or bilateral exophthalmos in 85%of cases.

Etiology :

- It is an autoimmune disease, which leads to formation of abnormal thyroid stimulating antibodies leading to stimulation of T.S.H. receptors on the thyroid cell membrane resulting in an action on the thyroid cell similar to T.S.H but prolonged.
- The disease is often precipitated by psychic trauma, emotional stress, acute infections, pregnancy or labor.

Pathology :

[1] Thyroid :

- The gland is highly vascular and friable.

Gross picture : There is diffuse small or large, smooth, soft or firm enlargement of thyroid gland.
Cut section : Homogenous, reddish brown & fleshy appearance.
Microscopic picture :

The acini	The stroma
1. Hyperplasia and hypertrophy. 2. The acini contain no or little colloid.	1. There is increased vascularity with A-V shunts. 2. Dense lymphocytic infiltration.

[2] On other organs : • There is hyperplasia of the R.E.S .

Clinical picture :

[A] Local manifestations :

1. Moderate, regular diffuse thyroid enlargement, with minimal pressure effects.
2. Smooth surface.
3. Soft consistency (less commonly firm).
4. Evidence of high vascularity :
(a) Systolic expansile pulsations.
(b) Thrill (on palpation) and bruit (on auscultation) maximum at upper poles, where superior thyroid arteries lie superficial.

[B] Systemic manifestations :

[1] Metabolic :

1. Loss of weight in spite of increased appetite.
2. Excessive sweating.
3. Intolerance to hot weather.
4. The hands of the patient are warm and moist.

[2] Nervous system :

Symptoms : Nervousness, insomnia or nightmares.
Signs :
- The patient looks tense and irritable.
- Tremors in the outstretched hands.
- Exaggerated reflexes.

[3] Cardiovascular system :

Symptoms :
- Palpitation.
- There may be anginal pains.
- Orthopnea, pain in the right hypochondrium and edema of the lower limbs (in case of HF).

Signs :
- Tachycardia with a sleeping pulse more than 90/minute.
- Water hammer pulse is due to high systolic (due to increased cardiac output) and low diastolic blood pressure (due to decreased peripheral resistance).
- **Arrhythmias :** all types of cardiac arrhythmias except heart block may occur. Arrhythmia is usually started by extrasystoles.

[4] Muscular :

- Muscular weakness with rapid exhaustion.
- In severe cases proximal myopathy and muscle atrophy may occur.

[5] Skin :

(a) Patchy pigmentation may occur.
(b) Pretibial myxedema :
- Presents as multiple orange yellow patches of thickening of the skin over the shin of the tibia.
- They are due to deposition of mucin like substances.

[6] Gastrointestinal tract : Diarrhea is due to increased c-AMP which increases the permeability of cells of mucous membranes.

[7] Endocrine system :

- **In fem ale :** Menstrual abnormalities.
- **In males :** Abnormal libido, gynecomastia or impotence.

[8] Urinary system : Polyuria and glycosuria.

[9] Eye manifestations :

Symptoms :

1. Blurred vision.
2. Diplopia.
3. Pain and photophobia (corneal ulcers).
4. Excessive tearing.
5. Swollen black eyelids.
6. Bulging.

Signs :

I- Tremors : Detected in lightly closed eyelids.

II- Exophthalmos : May be false or true.

[A] False exophthalmos :

- It accompanies both 1ry and 2ry toxic goitres.

Etiology : It is due to contraction of the levator palpebrae superioris (Muller's muscle) pulling the upper eyelid's tarsus and superior conjunctival fornix upwards.

- The high level of thyroid hormones sensitizes this muscle to the effect of circulating catecholamines.

Treatment : It usually disappears when the hyperthyroidism is treated.

[B] True exophthalmos :

- There is actual protrusion of the eyeball.

Etiology : Deposition of fat and round cell infiltrate in the retrobulbar tissues or it may be an autoimmune disease due to thyroid stimulating antibodies affecting the ocular muscles.

Results :

1. Ophthalmic vein compression, leading to edema, conjunctival congestion and chemosis.
2. Weakness of extra -ocular muscles results in diplopia.
3. In severe cases papilledema and corneal ulceration may occur. The severe progressive form is known as **malignant exophthalmos.**

Treatment :

- Usually self-limiting and may even regress.

1. Sleeping in the semi-sitting position and lateral tarsorrhaphy may help to protect the eyes.
2. High doses of systemic prednisone may be helpful.
3. Orbital decompression may be indicated when the eye is endangered.

N.B.: Hypothyroidism may increase the condition.

Clinical detection of exophthalmos

1. Naffziger's method :

- The examiner stands behind the patient, with the head tilted backwards.
- In true exophthalmos the eyeballs protrude beyond the plane of the superciliary ridge.

2. Russell Frazer's method :

- Examining the patient from the side with the eyes lightly closed, will determine the depth of the groove between the orbital margin and the covered globe (shallow in true exophthalmos).

3. Ruler test :

- Examiner uses ruler & tries to rest its edge in front of eyeball on the supraorbital edge & zygoma.
- Normally, it doesn't touch the cornea.

4. Exophthalmometer:measures the distance between lateral orbital margin and apex of the cornea:normally it is 15-17 mm.

III- Eye signs, other than exophthalmos, include :

1. Stellwag's sign : staring look with infrequent blinking (normal blinking 5-8 /min).

2. Von Grafes' sign : the upper eyelid lags behind the moving eyeball as the patient looks down without moving the head.

3. Dalrymple's sign : appearance of a rim of sclera between the upper eyelid and the cornea.

4. Joffroy's sign : lack of forehead wrinkling on looking upwards without moving the head.

5. Moebius sign : defective convergence due to muscle paresis (for 30 cm at least).

Thyrotoxicosis should be suspected in :

1. Children with growth spurt, behavioral problems or myopathy.
2. Elderly persons with unexplained tachycardia or arrhythmia (apathetic hyperthyroidism).
3. Unexplained diarrhea.
4. Unexplained loss of weight.
5. Resistant heart failure.

Differential diagnosis :

1. Primary thyrotoxicosis should be differentiated from secondary thyrotoxicosis :

Items	Primary thyrotoxicosis	Secondary thyrotoxicosis
1. On top of	Normal thyroid gland.	Simple nodular goitre.
2. Exophthalmos	True in 85% of cases.	Only apparent due to thyroxin.
3. Histology	Diffuse hyperplasia.	Hyperplasia of internodular tissue.
4. Age of onset	Younger (20-40).	Older (over 40).
5. Onset	Rapid, after stress.	Gradual.
6. Course	Fluctuating.	Progressive.
7. Degree	Usually severe.	Usually mild.
8. Main toxic presentation	Metabolic & nervous.	Cardiovascular, due to older age.
9. Local signs	Smooth regular goitre, small or moderate, with no pressure effects. High vascularity.	Nodular goitre may be huge. High vascularity.
10. Treatment	Drugs, surgery, radioiodine.	Surgery is the best.

2. Other causes of goitre.
3. Other causes of loss of weight in spite of increase of appetite e.g. parasitic infestations & D.M.
4. Anxiety neurosis.

Investigations :

- **Thyroid function tests T_3, T_4, TSH and ^{123}I uptake test** confirm the diagnosis.
- **T_3 thyrotoxicosis** is identified by elevated free T_3.
- **Toxic adenoma** shows as a hot nodule on ^{123}I scanning with suppression of the uptake of the surrounding thyroid tissue.

N.B: measurement of serum TSH: The test is routinely performed with normal range of 0.5-5 Mu/l. It is no possible to do **ultrasensitive TSH** and this test now is the most sensitive for assessment of thyroid functions.

Thyroid functions tests

[1] measurement of serum TSH

[2] Free T_3 & T_4 in the serum : (measured by radioimmunoassay)

Normal values :

- Free serum T_4 = 8 - 26 pmol/L.
- Free serum T_3 = 3-9 pmol/L (essential if T_3 thyrotoxicosis is suspected).

[3] Total T_3 & T_4 in the serum: (measured by radioimmunoassay).

Normal value:

- Total serum T_4 = 55 - 150 nmol/L.
- Total serum T_3 = 1.2 - 3.1 nmol/L.

[4] T.R.H. test :

- I.V. Thyrotropin releasing hormone.

Normal : Rise of T.S.H. level in the serum.

In thyrotoxicosis : No rise in T.S.H level in the serum.

[5] Thyroid antibodies: raised in Hashimoto's thyroiditis and Grave's disease.

[6] Radioactive iodine studies :

^{123}I is ideal as it has short half life (11 hrs.) while it is 8 days in ^{131}I.

Radioactive iodine studies include :

(A) ^{123}I uptake by thyroid gland and excretion in urine : (ordinary iodine is ^{127}I)

- 5 microcuries of ^{123}I is given orally → the uptake by the thyroid gland and the amount excreted in urine are measured at 4, 24 and 48 hours.
- In thyrotoxicosis : Very high dose of ^{123}I is taken rapidly by the thyroid gland with little excretion in urine → ^{123}I is discharged rapidly from the gland.

(B) Scanning of the thyroid gland :

- Using a special apparatus after giving a dose of $1^{123}I$, Technetium 99m or recently florescent scanning.

Values :

1. It gives an idea about the size and the shape of the gland.
2. Differentiate hot nodule i.e. increased activity (toxic) from a cold nodule i.e. decreased activity (cyst or malignant nodule).
3. Detect ectopic thyroid tissue e.g. thoracic or lingual.
4. Detect functioning thyroid metastasis.
5. It detects retrosternal extension.

[7] Blood examination :

- **In thyrotoxicosis there are :**

1. Hypercreatinemia : N: less than 1 mg%.
2. Hypocholesterolemia : N: 150 - 250 mg%.
3. Hyperglycemia : N: 120 mg%.

Treatment of thyrotoxicosis

[I] Non-specific measures : For all cases :

1. Rest. 2. Sedatives. 3. Nutritious diet and excess fluids.

[II] Specific measures to control thyrotoxicosis :

- Include anti-thyroid drugs, surgery and radioiodine.
- **Each patient is considered separately and one measure or a combination of measures is chosen for him.**
- **Choice depends on the following factors :**

1. Intelligence. 2. Socioeconomic factors. 3. The available medical facilities.
4. Age. 5. Pregnancy. 6. High thyroid antibodies titer.
7. General fitness. 8. Recent exophthalmos. 9. Cardiac complication (thyrocardiacs).
10. **Type of thyrotoxicosis :** examples :

(a) Primary toxic goiter : Surgery, anti-thyroid or radioiodine.
(b) Secondary toxic goiter : Surgery is always better because :
- Incomplete response to anti-thyroid drugs and radioiodine.
- Usually large with cosmetic and pressure effects and liable to all complications of nodular goitre.

(c) Toxic nodule : Surgery or radioiodine.
- Excision is easy and safe.
- Radioiodine is theoretically ideal, as suppressed thyroid tissue around the nodule will not take radioiodine, with no risk of delayed thyroid insufficiency.

(d) Recurrent thyrotoxicosis after adequate surgery : Radioiodine or anti-thyroid drugs.
(e) Failed anti-thyroid drugs or radioiodine : Surgery.
(f) Suspected malignancy : Surgery.
(g) Retrosternal goitre : Surgery, anti-thyroid drugs enlarge the goitre causing suffocation.

[A] Medical Treatment

Aim : to restore the patient to an euthyroid status, then to prescribe a maintenance dose for a prolonged period in the hope that a permanent remission occurs.

Indications :

1. Primary thyrotoxicosis. 2. Mild thyrotoxicosis.
3. Children and young patients. 4. Pre-operative preparation of patients.
5. Post-operative recurrence. 6. Refusal of surgery.
7. Small gland.

Contraindication :

1. Toxic nodular goitre. 2. Huge goitre causing pressure symptoms.
3. Retrosternal goitre. 4. Suspicious of malignancy.
5. Leucopenia or agranulocytosis. 6. Pregnancy and lactation.

Methods :

1. Sedative : ensure complete mental rest & good night sleep.
2. Inderal (B-blocker) : to decrease the HR and palpitation and partially inhibit conversion of T4 to T3.
Dose : 10 - 40 mg T.D.S. orally.
3. Anti-thyroid drugs :
Onset : Anti-thyroid drugs start to give their clinical effect after 2 weeks, thereafter it control thyrotoxicosis gradually till euthyroid state is reached.

Preparations :

[1] Lugol's iodine (5% I+ 10% KI) :

Indication : Used only in preoperative preparation.

Effect :

1. Reduces the effect of TSH on the thyroid gland and inhibits iodine binding.
2. Reduces the vascularity of the thyroid gland.
3. Leads to storage of colloid within the acini.
4. Its action declines after 2 weeks.

Dose : 10 drops tds with milk or juice to mask the bitter taste.

[2] Carbimazol : (The commonest anti-thyroid drug used)

- It blocks iodine binding to tyrosine and decreases antibody titers.

Dose :

- 10 mg tds (maximum dose 60 mg/d) till euthyroid state is reached then decrease the dose to 5 mg T.D.S for 12 - 18 months.
- If thyroxin 0.1 mg/day with anti-thyroid drugs → less danger of producing iatrogenic hypothyroidism or an increase in the size of the gland.

[3] Propylthiouracil :

- It blocks iodine binding to tyrosine and prevents peripheral conversion of T4 to the more active T3.

[4] Potassium perchlorate : It inhibits iodide trapping by the thyroid.

Advantages : Avoiding surgical risks and the possible hazards of radioactive materials.

Disadvantages :

1. There is no way to predict which patient is liable to pass into remission.
2. High relapse rate : 60% within 2 years from stopping treatment.
3. Further enlargement of the gland may occur.
4. Adverse effects of the drugs :
   - Thyouracil & Carbimazol may cause gastrointestinal upset, rashes & arthralgia.
   - Reversible bone marrow depression leading to agranulocytosis. Blood picture should be performed every two weeks.

[B] Radioactive iodine

Aim : It destroys the thyroid cells thus reducing the mass of functioning thyroid tissue.

Dose: 160 uCi per 1 gm of thyroid tissue. Improvement occurs by 8 - 12 weeks, if not a second dose may be required.

Indications :

1. Diffuse toxic goitre (above 25 y. old).
2. Thyrocardiac patients.
3. Refusal of surgery.
4. Recurrence after surgery.

Contraindication :

1. Below the age of 25 years.
2. During pregnancy & lactation.
3. Huge goiter, retrosternal goitre and suspicious of malignancy.

Advantage : Avoidance of surgery and prolonged medical therapy.

Disadvantage :

1. Thyroid insufficiency in up to 75-80% of cases after 10 years and so an indefinite follow-up is mandatory.
2. It is also inadequate for the treatment of secondary toxic goitre.

[C] Surgery

Aim : Surgery cures by reducing the mass of overactive tissues or removing all the overactive tissues in case of toxic nodule.

Indications of surgery are :
1. Secondary toxic goitre.
2. Severe primary thyrotoxicosis.
3. Retrosternal toxic goitre, huge goitre or suspicious of malignancy.
4. Failure of medical treatment.
5. Occurrence of side effects due to medical treatment.

Contraindications : Young patient, mild cases or recurrence after surgery.

Advantage : A rapid cure with a high rate of success.

Disadvantage :
1. Morbidity and mortality (negligible in experienced hands).
2. Recurrence rate less than 5%.
3. Thyroid insufficiency at an incidence of 20-45%.

Preoperative preparation :

[1] Routine Method :

Medical treatment :

- Neomercazole is given till the euthyroid state is reached then surgery is performed.
- Some surgeons add Lugol's iodine 10 - 14 days immediately prior to surgery to decrease the vascularity and friability of the gland.

[2] Rapid preparation :

By B-adrenergic blocker :

- The action of thyroid hormones especially on the CVS is proved to be through activation of adrenaline.
- Therefore inderal (propranolol) is now used for rapid control of CV manifestations before operation.
- It acts on target organs and not on the thyroid itself.
- So, it must be continued for one week after surgery.

Operation :

- The following precautions should be considered :

1. Pethidine and hyoscine are given before anesthesia (atropine is not used as it causes tachycardia).

2. The incision should be wide with division of pretracheal muscles, thus manipulation is minimal to avoid thyrotoxic crisis.

3. The operation done is **SubTotal Thyroidectomy** by which the main mass of the gland is removed leaving only a thin posteromedial wedge of each lobe to maintain an euthyroid state, preserve the parathyroids and protect the R.L.N. The amount of the gland left is 4-5 gm on each side.

4. Perfect hemostasis and free drainage to avoid thyrotoxic crisis.

Special problems in management

Thyrotoxicosis with pregnancy :

- **Radioiodine is absolutely contraindicated** because destruction of the fetal thyroid may result.
- **Antithyroid drugs** at usual doses would result in fetal hypothyroidism with the development of goitre that may obstruct the airway. Minimum dose antithyroid drug, preferably ß- blockers, reduces this risk.
- **Surgery after a short course of antithyroid drugs and propranolol** proved to be safe during the second trimester.

Thyrotoxicosis in children :

- **Radioiodine is contraindicated for children.**
- **Surgery is followed by a high recurrence rate** due to high activity of the cells in the young.
- It is preferable to treat them by **antithyroid drugs** until the late teens.

The thyrocardiac patient :

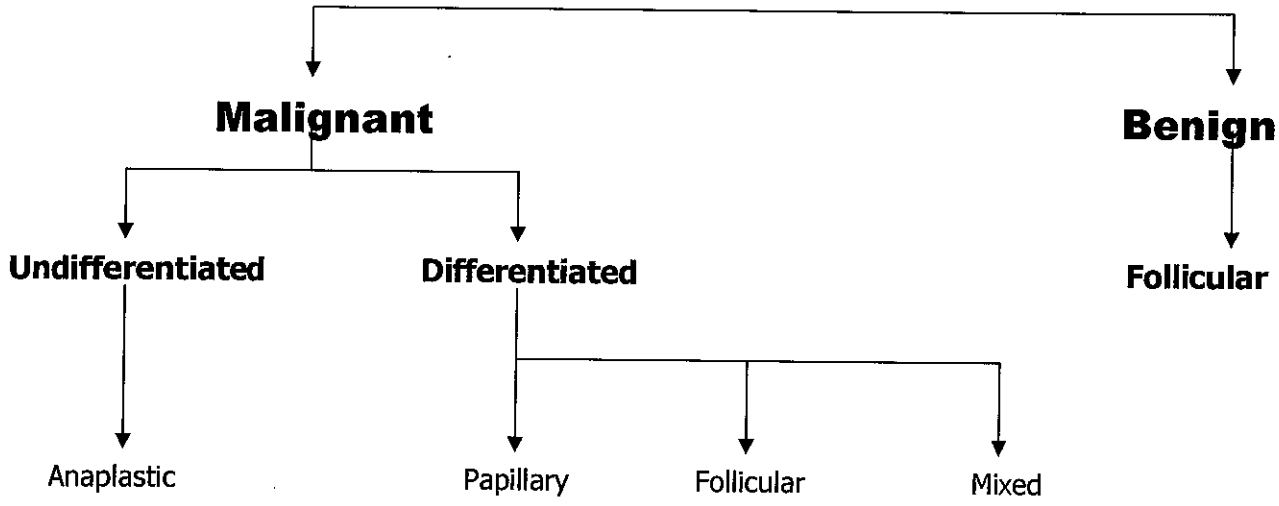
- **Thyroidectomy is ideal after control of the cardiac status.**
- **In unfit patients :** radioiodine is used with antithyroid drugs until the effect of radioiodine appears (6 weeks).

Proptosis of recent onset :

- It is not preferable to terminate the toxic status abruptly by surgery or radioiodine for fear of the risk of malignant exophthalmos.
- Antithyroid drugs are used until the proptosis has been stable for 6 months after which thyroidectomy is permissible.

Tumors Of The Thyroid Gland

1. Tumors arising from follicular epithelium :



2. Tumors arising from parafollicular epithelium : Medullary carcinoma.

3. Tumors arising from lymphoid elements : Malignant lymphoma

4. Rarely the thyroid gland is infiltrated by metastatic deposits or by local infiltration from a nearby lesion.

Follicular Adenoma

- Presents as a solitary nodule.
- Fine needle aspiration is not reliable to differentiate it from carcinoma as the main differentiating point in presence of histological evidence of capsular or vascular invasion.

Treatment :

Lobectomy plus isthmectomy.

(Many authors now consider any papillary adenoma as carcinoma).

Malignant Neoplasms

Incidence : More in females.

Predisposing factors :

1. External irradiation of the head and neck region in children : was previously used for treatment of hemangiomas and thymic gland enlargement.

2. Simple nodular goitre 3%.

3. Follicular carcinoma is the commonest type to develop in endemic goitrous areas : due to continuous stimulation by TSH.

4. Genetic : the lesion is more common in certain families. Hereditary factor is marked in medullary carcinoma.

5. Malignant lymphoma may occur on top of Hashimoto's disease.

6. Anaplastic cancer thyroid may occur DE NOVO.

Pathology :

[1] Tumors arising from follicular epithelium :

Items	Papillary	Follicular	Anaplastic
Incidence	60%	17%	13%
Age	May occur in children and young adults	Middle age	Elderly
Sex (F : M)	3.5 : 1	2 : 1	1 : 1.3
Microscopic picture	• Papillary projections composed of a stalk of connective tissue covered by a single layer of epithelial cells. • Laminated calcified bodies **(Psammoma bodies)** are often present in the stroma. The tumor shows characteristic pale empty nuclei **(orphan Annie-eyed nuclei)**.	• Thyroid follicles with a variable degree of differentiation. • Solid sheets may be present. • Diagnosis depends upon finding capsular or vascular invasion or the presence of metastases.	• It may take the form of spindle cell, small cell or giant cell types.
Multiplicity	Multiple foci may be present in the same or the other lobe in up to 80% of cases. These are due to either multicentricity or intrathyroidal lymphatic spread.	Rarely seen.	
Spread	Mainly by lymphatics	Mainly by blood stream	Early direct lymphatic or blood
10 year survival	90%	Encapsulated:97% Invasive :70%	Most of patients die within 1-2 years

N.B.: If the tumor contains both papillary and follicular elements it is considered papillary

[2] Tumors arising from parafollicular cells : (Medullary carcinoma 6%)

• The tumor may occur **alone**, or it may be associated with hyperparathyroidism and pheochromocytoma constituting **(MEN IIa).**

• **The tumor may be familial** and in this case, it is more common in children and young adults.
• **It secretes calcitonin** (Tumor marker).
• **Microscopically :** Sheets of neoplastic cells in a hyaline stroma, which may contain amyloid material.
• **Spread :** Lymphatic or blood.
• **There is diarrhea in 30% of cases.**

MEN (multiple endocrinal neoplasia) syndrome

Inherited condition, autosomal dominant, all family members should be screened.

[1] MEN1 (Wermer's syndrome) :

1. **P**arathyroid Hyperplasia
2. Islet cell tumors of **p**ancreas (e.g. gastrinoma).
3. **P**ituitary tumor.

[2] Type II Sipple's syndrome :

• **Type IIa :**

1. Parathyroid hyperplasia.
2. Pheochromocytoma.
3. Medullary Carcinoma (thyroid).

• **Type IIb:** Pheochromocytoma, Medullary Carcinoma and mucosal neuromas.

[3] Lymphoma.

TNM staging

T1: limited to thyroid gland, 1cm or less
T2: limited to thyroid gland, 1-4 cm.
T3: limited to thyroid gland,> 4 cm.
T4: extended beyond the capsule

N0 no nodal metastasis **M0** no distant metastasis
N1 nodal metastasis **M1** distant metastasis

N.B: microcarcinoma is a term used for cancers less than 1 cm in diameter. they carry excellent prognosis.

Clinical Picture :

- May be history of irradiation.

[A] Early or Suspicious presentations :

1. If on top of pre-existing goiter :
(a) Faster growth rate.
(b) Harder consistency.

2. If a lump appears de novo : The rule is that any single thyroid nodule (especially in young males) is considered malignant until proved otherwise, especially if rapidly growing, hard, irregular and ill defined, considering that carcinoma can occur at any age from 15 to over 60y.

3. Rarely, the patient presents with metastases while the primary is impalpable. This occurs particularly in papillary type and the patient presents with enlarged deep cervical nodes. In the past, before knowing the nature of such nodes they were called **lateral aberrant thyroid.**

[B] Late (sure) presentations : Goitre with spread

I- Local spread :

1. Hoarseness of voice (infiltrated RLN). However, laryngitis, tracheitis and RLN damage by past fever or previous thyroid surgery should be excluded.
2. Pain radiating to ear (infiltrated vagal thyroid branches). However hemorrhage or infection in a nodule should be excluded.

3. Dysphagia (infiltrated esophagus). Rarely it occurs in retrosternal simple goitre.
4. Severe dyspnea (infiltrated trachea).

5. Fixity to skin, sternomastoid or trachea.
6. Surrounding carotid sheath, biding carotid pulse instead of pushing it backwards **(Berry's sign).**

II- Lymph node metastasis :

- Mainly in prelaryngeal, pretracheal and lower deep cervical nodes.
- First lymph node affected in thyroid carcinoma is RLN then pretracheal and prelaryngeal (Delphic).

III- Blood metastasis :

N.B bone metastases present by: Painful, tender, pulsatile lump or pathological fractures (Common sites are spine, skull & neck of femur).

N.B.: Medullary carcinoma produces diarrhea in 30% of cases due to production of 5-hydroxytryptamine or prostaglandins.

D.D :Other causes of goitre especially hard nodules e.g. Tense cyst, calcified nodule & Reidel's thyroiditis.

Investigations :

[A] Laboratory Test : (For Tumor markers)

1. Serum levels of thyroglobulin : It is elevated in carcinoma as well as other types of goitres. It is only useful for following patients after total thyroidectomy to detect metastasis or recurrence.

2. Detection of serum calcitonin : elevation in medullary carcinoma.

[B] Radiological :

1. Thyroid scanning (of limited diagnostic significance). Malignant lesion usually appears as cold areas in the scan, but it should be remembered that many benign lesions as cysts or hemorrhage in a cyst or degenerative nodules or thyroiditis appear also as cold areas.

2. Ultrasound examination : can prove whether the lesion is cystic, solid or cystic with solid projection.

3. C.T. & M.R.I.

[C] Aspiration of cyst :

A benign cyst is characterized by :	A cystic malignant lesion is suspected when :
The aspirate is clear.	The aspirate is hemorrhagic.
Complete disappearance of the cyst.	A residual lump is present.
No reaccumulation of fluid.	Rapid reaccumulation of fluid in the cyst.
Cytological examination is free.	Cytological examination may be positive for malignant cells.

[D] Biopsy :

1. Fine needle aspiration cytology (FNAC) :

- An experienced pathologist can give accurate results in 90% of cases.
- This is a simple, fast, inexpensive investigation, which depends upon cytological examination of nodules 0.5 cm or more.
- False positive results occur in 2% while false negative results occur in 3% of cases.
- In follicular carcinoma, FNAC may not be conclusive as the diagnosis of this lesion depends upon demonstration of capsular or blood vessel invasion.

2. Pre-operative L.N. biopsy : may be done from an enlarged cervical node.

3. Frozen section examination during open operation.

4. Excision biopsy (hemithyroidectomy).

5. Incision biopsy is absolutely contraindicated as it results in seedling of malignant cells.

[E] Investigations to detect spread :

1. Chest X-ray : to detect pulmonary deposits.

2. Laryngoscopy : to check the mobility of the vocal cords.

Treatment :

I- Differentiated carcinoma

[1] Surgical treatment :

- In operable and if possible in inoperable cases (as palliation) either total thyroidectomy or lobectomy plus isthmectomy.

- **Surgeons who perform total thyroidectomy favor this procedure because :**

1. There is a high incidence of muiticentric lesions in papillary carcinoma.
2. Total thyroidectomy ablates all thyroid tissue and makes it possible to detect metastasis by scanning and to treat them by radioactive iodine.
3. It is easy to assess recurrence by serum thyroglobin.

- **Surgeons who perform lobectomy and isthmectomy favor this procedure because :**

1. No statistical difference was shown between the results of the two procedures.
2. Total thyroidectomy has a high incidence of post-operative hypoparathyroidism.

- **Management of lymph node metastases : (only in operable cases)**
  - If there are no palpable nodes: only the central lymph nodes are excised.
  - If there are metastasis in the posterior triangle: modified block dissection is done(spinal accessory nerve, internal jugular vein and sternomastoid muscle are preserved).

[2] Postoperatively L-thyroxin :

- 0.1-0.2 mg/day is prescribed to suppress TSH production, as most differentiated carcinomas are TSH dependent.

[3] Management of blood borne metastasis :

- Thyroxin is stopped for few weeks or substituted by tri-iodothyronine (to raise TSH) and then iodine scan is performed.
- If there is evidence of local recurrence or metastases, a therapeutic dose of radioactive iodine is prescribed.

II- Treatment of medullary carcinoma

- This tumor does not concentrate radioactive iodine and is not under the control of TSH.
- Treatment is by total thyroidectomy and resection of metastatic lymph nodes.

III- Treatment of anaplastic carcinoma

- The majority of these patients are inoperable when first seen.
- Rarely the lesion is operable and an attempt at cure by total thyroidectomy,
- The usual treatment is external irradiation and chemotherapy. If there is tracheal compression, resection of the isthmus is performed

VI- Treatment of lymphoma

1. Radiotherapy & chemotherapy.
2. Surgery only :
   (a) To establish the diagnosis.
   (b) Relieve a compressed airway.

Features of inoperability

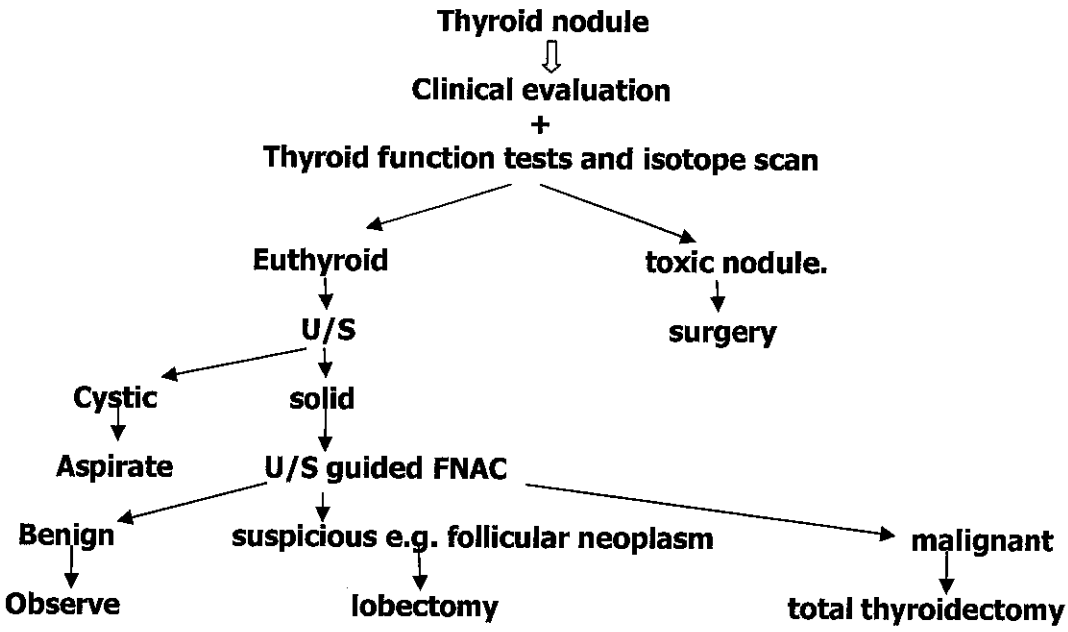
1. Infiltration of vital structures as carotids, trachea, bone etc.
2. Blood spread.
3. Adherent L.N. to vital structures.
4. Unfit patient
5. Anaplastic carcinoma is usually inoperable.

Prognosis of differentiated thyroid carcinoma

Certain factors increase the risk including :

1. **Age of the patient :** Males above 40 and females above 50 years of age.
2. **Sex :** Females has better prognosis.
3. **Size of the lesion :** If more than 5 cm in diameter.
4. **Presence of capsular or blood vessel invasion.**
5. **Presence of distant metastases.**
6. **Histological type.**

Algorithm for the management of a solitary thyroid nodule



Solitary Thyroid Nodule

Definition :

A goitre, which on clinical examination appears to be a single nodule in an otherwise normal gland.

Incidence :

More common in females and the commonest site is at the junction of the isthmus with the lateral lobe.

Etiology :

1. Simple nodular goitre with one palpable nodule (the commonest).
2. Toxic nodule.
3. Colloid nodule.
4. Adenoma.
5. Malignant nodule.
6. Inflammatory nodule (thyroiditis).

Clinical picture :

[1] Malignant manifestations : (In case of malignant nodules)

How to suspect malignancy in a solitary nodule ?

1. History of previous irradiation.
2. Young & elderly patients.
3. Recent onset & rapid growth.
4. Pain.
5. If the nodule is hard, irregular, with limited mobility.
6. Presence of local invasion, lymphatic or blood born metastases.

[2] Toxic manifestations : (In case of toxic nodules)

[3] Cases which are not Frankly malignant or toxic : The case may be :

1. Early malignant nodule.
2. Early toxic.
3. Simple nodule.
4. Adenoma.
5. Inflammatory nodules.

- **In these cases, investigations are essential to diagnose the case.**

Investigations :

1. Thyroid scan :

- May reveal the presence of multinodular goitre.
- If the nodule is hot, it is toxic and the possibility of malignancy is nearly excluded.
- If the nodule is warm, it is a functioning adenoma and the possibility of malignancy in only 3.5%.
- If the nodule is cold, the possibility of malignancy is 10-16%.

2. Ultrasound.

3. Biopsy (see carcinoma).

4. Aspiration.

5. Thyroid function test : Not routine.

6. Investigation of carcinoma : Not routine.

Treatment : Depend on the cause :

1. Malignant nodule: see before.

2. Toxic nodule : see before.

3. Cases which are not frankly malignant or toxic :

- Hemithyroidectomy and histological examination i.e. excision biopsy.
  (a) If not malignant → nothing more is needed.
  (b) If malignant → see treatment of thyroid carcinoma.

Thyroiditis

I- Acute Thyroiditis :

- The thyroid gland is rarely involved by bacterial infection.

Clinical picture : Sudden onset of severe neck pain with dysphagia, fever.
Treatment : antimicrobial drugs, however, surgical drainage of suppuration may be required.

II- Subacute Thyroiditis : (Granulomatous thyroiditis, De Quervain Thyroiditis)

Etiology : Most probably a viral infection (usually self-limited).
Clinical picture :

- Usually follows an upper respiratory infection.
- The thyroid becomes congested, swollen, firm, irregular & slightly tender.
- Mild manifestations of thyrotoxicosis may occur due to libration of hormones from destroyed tissue
- Pain with fever.

Rapid response to oral prednisolone is diagnostic

Investigations :

- Elevation of ESR with a normal or depressed leucocytic count.
- Iodine-uptake by the gland is depressed in the presence of a slight elevation of serum T^4.

Treatment : Oral prednisone & anti-inflammatory may be used.

III- Chronic Thyroiditis : due to tuberculosis or syphilis is rare.

Autoimmune thyroiditis (Hashimoto's Thyroiditis)

Incidence :

- The most common form of thyroiditis.
- Usually affects females at menopause

Pathologically :

- There is infiltration of the thyroid gland by lymphocytes and plasma cells.

Clinical picture :

- **Onset :** Insidious and asymptomatic or sudden and painful in others.
- The goitre usually lobulated, diffuse or localized to one lobe, soft or firm, flat topped nodularity.
- Mild hyperthyroidism may be present initially but hypothyroidism is inevitable.

Investigation :

- Serum titers of antimicrosomal and anti-thyroglobulin antibodies are elevated
- Biopsy. *FNAC or true cut biopsy may be required to differentiate it from nodular goiter or carcinoma*

Treatment :

1. Full replacement dosage of thyroxin.
2. Surgical treatment : Indications :

- Large-sized goitres.
- Suspected development of papillary carcinoma, lymphoma or both.

Riedel's Thyroiditis

- 0.5% of goitres.
- The thyroid is hard due to extensive fibrosis, which extends even beyond the gland.
- It is probably a collagen disease.
- Hypothyroidism is usually present.
- The differentiation from anaplastic carcinoma may need an open biopsy where a wedge of the isthmus is removed to free the trachea.

N.B:thyroid incidentinoma:
It is clinically unsuspected and impalpable thyroid swelling.

Thyroidectomy

Indications :

1. Unilateral lobectomy :
(a) All solitary nodules.
(b) Multinodular disease in one lobe only.

2. Subtotal thyroidectomy :
(a) Diffuse toxic goitre.
(b) Bilateral toxic multinodular goitre.
(c) Bilateral simple multinodular goitre.

3. Total thyroidectomy : Papillary, follicular & medullary carcinoma.

4. Isthmectomy :
(a) Anaplastic carcinoma & lymphoma to free airway & obtain tissue diagnosis.
(b) Riedel's thyroiditis.
(c) Nodule in the isthmus.

5. Near total thyroidectomy=lobectomy+isthmectomy+near total lobectomy:

Complications after thyroidectomy :

• Apart from general surgical complications (shock, hemorrhage, infection, pulmonary complications and deep vein thrombosis).

[1] Post-operative thyroid crisis (Storm) :

> This is a condition of **"acute hyperthyroidism"**

Incidence : Rare nowadays due to the good preparation & control of toxicity before operation.

• It can occur during the course of the disease excited by severe nervous disturbance or a minor operation e.g. extraction of a tooth or incision of an abscess.

• More commonly it occurs after subtotal thyroidectomy. It usually starts within the 1st few hours to 48 hours after the operation.

Manifestations :

1. Hyperpyrexia : Temperature may rise to 41 °C or more.
2. Rapidly rising pulse rate : 160 or more, may be irregular.
3. BP rises greatly, systolic & diastolic (e.g. 250/120). The pulse pressure is high, so HF may occur.
4. Muscular excitability up to convulsion.
5. Dyspnea due to lack of O_2 as the metabolic rate is greatly increased.

Fate : The condition, if severe, and if left untreated, death occurs in short time.

Treatment : Should be urgent :

1. Ice packs to the limbs, head & abdomen. In severe cases the whole patient may be immersed in cold water or ice.
2. ß-adrenergic blockers (Inderal) : I.V. drip until the pulse drops to about normal should be given carefully under ECG control.
3. Morphia for sedation.
4. O_2 inhalation is essential & I.V. hydrocortisone (25mg).

[2] Reactionary hemorrhage :

• Results from continuous venous ooze following bad hemostasis. This hemorrhage is serious because it collects under deep fascia, compressing trachea. Suffocation may occur.

Management :

1. Immediate reopening of the wound.
2. Then transferring patient to theatre to explore the operation field under anesthesia and ligate bleeders.

[3] Dyspnea : May be due to :
1. Reactionary hemorrhage.
2. Bilateral incomplete RLN injury = Spasm of both cords = Severe dyspnea= Urgent tracheostomy (immediately after extubation).
3. Laryngeal edema from rough intubation (= tracheostomy).
4. Tracheal collapse from tracheomalacia (= tracheostomy).
5. Thyrotoxic heart failure (propranolol - digitalis).

[4] Change of voice :

1	Unilateral RLN injury	Hoarseness
2	Bilateral complete RLN injury	Aphonia
3	External laryngeal nerve injury	Loss of high-pitched voice
4	Tracheitis and laryngitis	May cause temporary change in voice

[5] Injury of nerves related to thyroid :

Effects of RLN injury (on voice and respiration)			
1	Unilateral complete injury	Paralysis of one cord	Hoarseness + Dyspnea on exertion
2	Unilateral incomplete injury	Spasm of one cord	Hoarseness + Dyspnea on exertion
3	Bilateral complete injury	Paralysis of both cords	Aphonia + Dyspnea on exertion
4	Bilateral incomplete injury	Spasm of both cords	Aphonia + Stridor

- 3% of people have RLN paralysis (without operation) may be due to viral infection → neuritis. So do preoperative laryngoscopy (medico-legal).

[6] Tracheitis and laryngitis = Temporary change of voice + dyspnea.

[7] Surgical emphysema = Tracheal injury = Immediate suturing.

[8] Hypoparathyroidism :
- Rarely occurs if all parathyroid are removed, or their vessels injured.
- Cramps (tetany) start few days after operation.

Emergency treatment : 20 ml of 10% calcium gluconate IV slowly.
Maintenance treatment : Ca + Vit D.

[9] Progressive exophthalmos :
Management :
1. Thyroxin to inhibit pituitary.
2. Preventing progression by sleeping semi-sitting and cortisone.
3. The eye is protected by :
   (a) Tarsorrhaphy.
   (b) Orbital decompression in malignant exophthalmos.

[10] Hypothyroidism : If enough thyroid tissue is not left.

[11] Recurrence : Of toxic or nodular goitre.

[12] Keloid in the scar :
Occurs in low wounds on sternum. It is avoided by placing the incision in the lower neck crease.

Hyperparathyroidism

Types :

1. Primary hyperparathyroidism : Autonomous production of parathormone with loss of feedback mechanism.

2. Secondary hyperparathyroidism : Due to compensatory hypersecretion of parathormone secondary to low serum calcium.

3. Tertiary hyperparathyroidism : Starts as secondary type but develops autonomous dysfunction.

Primary hyperparathyroidism

Incidence :

- 0.1-0.3 % of the general population.
- More in females, particularly in the 5th & 6th decades of life.
- Familial predisposition & sometimes a part of multiple endocrine neoplasia (MEN).

Etiology :

1. Single adenoma (92%).
2. Multiple adenomata (4%).
3. Hyperplasia (3%).
4. Parathyroid carcinoma (1%).
5. Rarely ectopic PTH producing cancers, especially that of lung, kidney & bladder.

Clinical presentation :

- Usually detected by laboratory investigations.
- Patients below 60 years present by urological manifestations.
- Patients above 60 years present by neuromuscular & psychiatric manifestations.
- In general 80% present with renal involvement, 35% skeletal involvement.

1. The earliest complaints : muscle weakness, anorexia, nausea, constipation, polyuria, & polydipsia.

2. Renal presentation :

- Nephrolithasis (30_80%).
- Nephrocalcinosis (5_10%), irreversible & may lead to renal failure & hypertension.
- Renal stones with its presentation.

3. Bone diseases : Subperiosteal resorption especially of the phalanges & in severe cases bone cysts.

4. GIT manifestations : Peptic ulcer.

5. Emotional disturbances.

6. Articular & soft tissue manifestations : Chondrocalcinosis (3_7%).

7. Hyperparathyroid crisis : In severe hypercalcemia (16_20mg/dl). It presents by rapidly developing muscular weakness, nausea, vomiting, weight loss, fatigue & drowsiness.

D.D of hypercalcemia :

1. Primary hyperparathyroidism.

2. Malignancies :

- Group I (30%) : Hematologic malignancies as multiple myeloma & lymphomas.
- Group II (50%) : Solid tumors with lytic bone metastasis as cancers of lung, kidney & pancreas.
- Group III (20%) : Ectopic PTH producing cancers, especially that of lung, kidney & bladder.

3. Others :

- Milk-alkali syndrome.
- Hyperthyroidism.
- Paget's disease of bone.
- Vitamin D intoxication.
- Sarcoidosis.

Laboratory diagnosis :

1. Serum calcium : Total level is elevated except in cases of severe hypoalbuminemia.
2. PTH essay : Elevated level but in presence of hypercalcemia only.
3. Serum chloride to phosphate ratio above 33 suggests hyperthyroidism.
4. Increased urinary calcium 24 hours.
5. Increased serum alkaline phosphatase with skeletal lesions.

Localization :

- The most accurate method is neck surgical exploration.
- Preoperative methods include :

1. High resolution U/S with 76% accuracy.
2. CT scan has 50% accuracy & is helpful in medistinal lesion.
3. Thalium-Technetium scan (thyroid-parathyroid subtraction scan) : 72% accuracy.
4. In patients with previous neck exploration, **selective venous sampling** at certain points along big veins for serum PTH.

Treatment :

- **Treatment is surgical.**

1. Parathyroid adenoma : The adenoma is excised. Operative localization can be done by giving radioactive technetium & tracing the highest radiation density in the neck by a hand–held probe. The others are exposed to ensure that they are of normal size. One of them is biopsied.

2. Parathyroid hyperplasia :
(a) Subtotal thyroidectomy : i.e. excision of three & half glands.
(b) Other alternative include : total parathyroidectomy with heterotropic auto-transplantation of very thin slices in the forearm muscles. Postoperative hypercalcemia requires removal of few parathyroid slices.

- 80% of parathyroid tumors are attached to the posterior capsule of thyroid gland, symmetrically placed & overlie the recurrent laryngeal nerve.
- The superior glands are placed far dorsally at level of upper 2/3 of thyroid lobes.
- The tissues inferior to thyroid lobe are cleaned down to trachea to expose recurrent laryngeal nerve.
- It is helpful to follow the branches of the inferior thyroid artery especially if one is enlarged.
- If both lower glands cannot be found, the thymus pedicle should be carefully examined.
- The glands should be gently handed as their blood supply is easily damaged & the field should be bloodless to allow for search.
- Postoperative care : serum calcium should fall to normal within 48 hours. If hypocalcemia occurs, oral calcium supplementation or even IV calcium can be administrated.

Secondary & tertiary hyperparathyroidism

Secondary hyperparathyroidism : Occurs in :
1. Renal osteodystrophy.
2. Malabsorption syndrome.
3. Rickets & osteomalacia with low serum phosphorus.

Tertiary hyperparathyroidism :

- Prolonged chief cell hyperplasia cause them to be autonomus with elevated serum PTH & calcium.

Treatment of both : Essentially medical :
1. Vitamin D.
2. Calcium & phosphate binders.
3. Surgery in failure response to medical treatment by removal of 50 gm of parathyroid or 15 slices 1mm each.

Neck

Neck

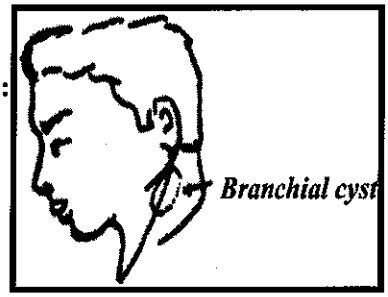
Anatomical divisions of the neck

- **Triangles**
- **Compartments :** *the pre-vertebral fascia divides the neck into 2 Compartment*
  - **The muscular Compartment**
  - **The visceral Compartment**

Branchial cyst

Etiology: Origin:

- ***In embryo,*** there are **5** grooves on each side of neck called branchial clefts (like fish gills).
  - ✓ First cleft persists as external auditory meatus.
  - ✓ Other 4 clefts disappear normally.



Second cleft is the commonest to persist and produce 2 anomalies:

I. **Branchial fistula :** if cleft persists from skin to pharynx.
II. **Branchial cyst :** if mid-part of cleft is sequestrated.

Incidence: Congenital but presents in childhood or later.

Pathology:

Lining: Squamous epithelium, surrounded by lymphoid tissue, which explains its frequent inflammation
Content: Mucus rich in cholesterol crystals (rectangular with notched corner).

Clinical Picture:

Site :

- Upper part of side of neck
  below angle of mandible deep to upper 1/3 of sternomastoid protruding beneath its anterior border
- On contracting sternomastoid, branchial cyst seems to bulge out.

Characters :

- Usually starts after the age of 20 years.
- Moderate size (5 cm diameter), smooth, globular, tense cyst, well defined and opaque.

DD: From other swellings in the lateral aspect of the neck.
Branchial cyst is differentiated from cold abscess by finding cholesterol crystals in the aspirate

Complications: Frequent inflammation as its lining is surrounded by lymphoid tissue.

N.B: It is always connected to pharynx :

- Usually by fibrous band passing between external and internal carotids to end high in lateral wall of pharynx behind tonsil **(Supratonsillar fossa of Rusenmuller).**
- Rarely by open duct.

Treatment :

- **Complete excision.** Through transverse incision, cyst is dissected and its fibrous band followed between carotids to lateral wall of pharynx.

Branchial fistula

[A] Usually congenital : Present since birth.

Clinically:

1. External opening :

- Lies on lower 1/3 of sternomastoid near its anterior border.
- It discharges mucus, and if infected pus.
- Fistula is felt as thread passing up and deeply through anterior part of sternomastoid.

2. Internal opening :

- Fistula passes between external and internal carotids to end high in lateral wall of pharynx behind tonsil, usually blindly or rarely opening into it.

Lining: Squamous epithelium, surrounded by lymphoid tissue, which explains its frequent inflammation (as pharynx)

D.D. : T.B. sinus.

Treatment :

Complete excision

- Transverse elliptical incision is made around external opening fistula is followed between carotids to lateral wall of pharynx.
- Higher skin incision is added to facilitate dissection **(step-ladder incision)**.
- A ureteric catheter may be introduced into the track to facilitate the operation.

[B] Sometimes branchial fistula is acquired:

- ✓ Due to rupture or incision of inflamed branchial cyst or incomplete removal of cyst

Clinically, it differs from congenital fistula in :

- ✓ **Age :** It appears in *adulthood*, on top of pre-existing cyst.
- ✓ **Site :** External opening lies *high* on side of neck (cyst level)
- ✓ **Treatment :** complete surgical excision.

Cystic hygroma (Cavernous lymphangioma)

- **Not true tumor, but tumor-like congenital malformation (hamartoma)**
- **consisting of irregular lymph spaces, with no capsule.**

Origin : Sequestrated part of jugular lymph sac of fetus.

Incidence : Rare, Appears at birth or shortly after.

Pathology :

- It consists of multiple intercommunicating cystic spaces like soap bubbles.
- Cysts near surface are large while deeper ones small and infiltrate muscles.
- It is lined by endothelium and contains lymph.

Clinically :

- Commonest in lower part of side of neck, superficial to sternomastoid and extends to posterior triangle.
- It may enlarge upwards and reach ear.
- It is large, ill defined, irregular, lax, cystic, lobulated, translucent (All other neck swellings are opaque).
- Partially compressible due to shift of lymph to deep cysts, and expands if child cries or coughs.
- It may contain solid areas due to clotting and fibrosis.

D.D.:

Other causes of cystic swelling in lateral side of the neck.

Items	Branchial cyst	Cystic hygroma
Origin	Sequestrated part of branchial cleft.	Sequestrated part of Jugular lymph sac.
Composition	Single cyst.	Intercommunicating cysts.
Lining	Squamous epithelium.	Endothelium.
Content	Mucus + cholesterol crystals.	Lymph .
Age of onset	Child or adult.	Infant.
Site	High in neck, deep to sternomastoid.	Low in neck, superficial to sternomastoid.
Shape	Globular.	Irregular.
Size	Moderate (5 cm diameter).	Large.
Surface	Smooth.	Lobulated.
Consistency	Tense cystic.	Lax cystic.
Edge	Well-defined.	Ill-defined.
Transillumination	Opaque.	Translucent.

Other sites :

- Second common site is axilla, affected alone or with neck.
- Rare in cheek, lip **(macrocheilia),** tongue **(macroglossia).**
- Very rare in groin, mediastinum or pelvis.

Fate :

- Usually continues to enlarge. In neck, this may cause suffocation.
- Rarely spontaneous regression occurs after infection.

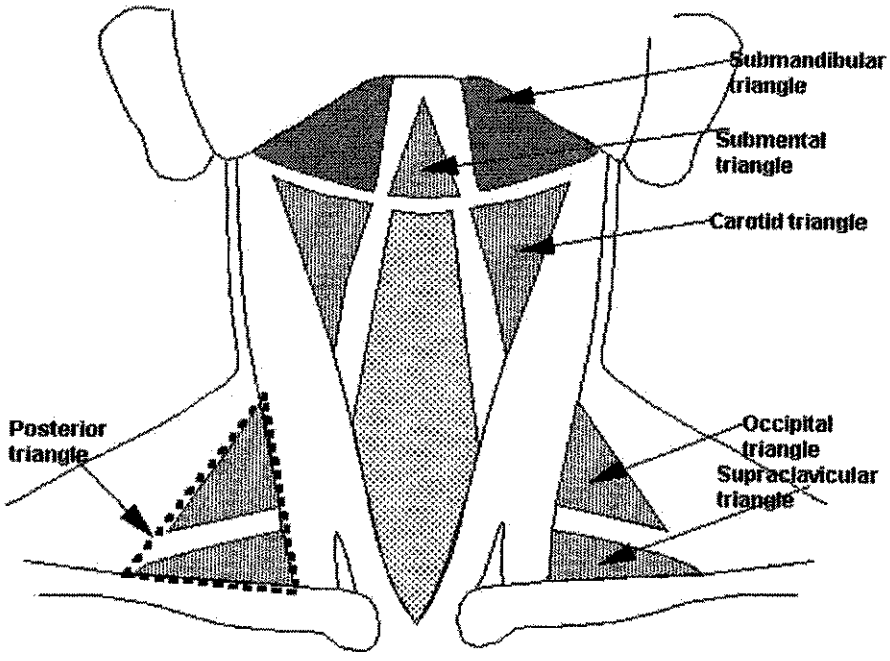
Treatment :

Excision as early as possible. Dissection may be facilitated by preliminary injection of boiling water or sclerosing material weekly for several weeks to induce fibrosis.

✓ Excision of at the age of 3 years should be done.

Differential diagnosis of a mass in the neck

- It should be noted that the most common swellings of the neck are swellings of lymph nodes and thyroid, followed by those of the salivary glands.



Mid line swellings

Solid swellings :

1. **Submental lymph node enlargement.**
2. **Nodule in the isthmus of the thyroid gland.**

Cystic swellings :

1. **Cold abscess**
2. **Dermoid cyst :** Sublingual or suprasternal.
3. **Subhyoid bursitis :** It is a rare, tender, oval swelling which lies transversely beneath the hyoid bone. It moves up and down with deglutition and with protrusion of the tongue.
4. **Laryngocele :** It occurs in musicians playing with air-blown instruments.
   It is a herniation of laryngeal mucosa through the thyrohyoid membrane.
   The swelling is resonant, compressible & increases in size with coughing or blowing.
5. **Cystadenoma of the thyroid isthmus**

Swellings in the digastric triangle

1. **Enlarged submandibular lymph nodes :** multiple, can be rolled over the edge of the mandible and can't be felt in the floor of the mouth.
2. **Enlarged submandibular salivary gland.**

Swellings in the carotid triangle

Solid swellings :

1. **Enlarged upper deep cervical lymph nodes.**
2. **The upper part of an enlarged lateral lobe of the thyroid gland.**
3. **Carotid body tumor :** It is a rare slowly growing malignant tumor arising from the chemoreceptors present at the bifurcation of the carotid artery. The swelling moves from side to side but not vertically. It may be pulsating. Treatment by excision of the tumor with preservation of the internal carotid artery.
   - *If preservation of the internal carotid artery is impossible, it should be replaced by a graft.*

Cystic swellings :

1. **Cold abscess.** 2. **Branchial cyst.**
3. **Aneurysm of the carotid artery.**

Swellings in the posterior triangle

Solid swellings :

1. **Enlarged lymph nodes.**
2. **Neurofibroma arising from the brachial plexus.**
3. **Cervical rib.**

Cystic swellings :

1. **Cystic hygroma** 2. **Pharyngeal pouch** 3. **Cold abscess**
4. **Pneumatocele :** This is a cystic swelling in the supraclavicular region, which is resonant and compressible. It is due to herniation of the pleura in the base of the neck.

- In addition, it should be noted that swellings of the skin and subcutaneous tissue are common in the neck region and should be put in mind such as **lipoma, sebaceous cysts,** and **hemangiomas,** which are particularly common in the neck and face regions.

Neck swellings with expansile impulse on cough & compressible :

- Laryngocele, Pharyngeal pouch & Pneumatocele.

Sternomastoid tumor and congenital torticollis

Etiology : Abnormal development

Interruption of the blood supply to the central somite of sternomastoid muscle. This causes muscle infarction which becomes swollen, hence the name sternomastoid tumor. After a while the infracted part will be replaced by fibrous tissue that will contract causing congenital torticollis.

Clinical features :

- At birth (or shortly afterwards) there is a painless firm swelling at the middle of sternomastoid muscle.
- Later when torticollis develops the head will be tilted to the side of the lesion with the face looking to the opposite side.
- If the condition is neglected it leads to facial asymmetry.

Differential diagnosis : from **'wry neck'** which is fibrositis causing spasm of the sternomastoid muscle. This condition is temporary lasting for a day or two and responds to anti-inflammatory drugs.

Treatment :

- **Early after birth :** Attempt to prevent the development of the deformity by stretching and splinting of the neck may be done.
- **Established deformity :** Division of the sternomastoid at its lower part is done.

Neck injuries

Etiology : Blunt & Penetrating trauma.

Pathology :

- Blunt trauma usually causes fracture of the spine and only in severe injuries it may be associated with visceral and vessel injuries.
- Penetrating injuries deep to the platysma are usually associated with injury to viscera and vessels.

Complications :

1. Fracture of the cervical spine can lead to quadriplegia or paraplegia.
2. Injury to the larynx can lead to respiratory obstruction, aphonia or hoarseness of voice. Laryngeal stenosis may be a late complication.
3. Arterial injury leads to hemorrhage which may be severe enough to cause shock. It may also lead to interruption of the blood supply to the brain causing infarction and a neurological deficit.
4. A vein injury in addition to hemorrhage can lead to air embolism.
5. Injury to the esophagus will be followed by leakage and severe infection in the neck which may extend to the mediastinum.

Clinical features : Of complications.

Investigations :

Plain x-ray and angiography are very useful to detect fractures and vessel injury. However, in cases with severe bleeding, immediate exploration is necessary.

Treatment :

Life-saving measures : primary and secondary surveys.

Definitive treatment :

- Visceral and vascular injuries : Exploration and repair.
- Treatment of spinal injuries.

Cellulites of the neck

Superficial cellulites

Deep cellulites (Ludwig's angina)

✓ Ludwig's cellulitis is usually results from streptococcal infection.

Pathology :

- It is a clinical condition characterized by inflammatory swelling of both the submandibular region and the mouth cavity
- Infection is deep to the deep fascia and can spread to the glottis causing edema and asphyxia

Clinical features :

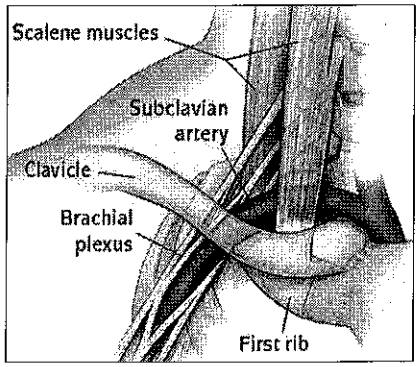
- Painful, red, warm, tender submandibular swelling
- The overlaying skin is edematous

Treatment :

Antibiotics and drainage through an incision beneath the jaw

Thoracic outlet syndrome

- **The brachial plexus** and **the subclavian artery** pass to the upper limb through a narrow triangle in the base of the neck.
- This triangle is made up of the scalenus anterior muscle anteriorly, the scalenus medius muscle posteriorly and the first rib inferiorly.
- At this narrow space compression of nerves and artery may occur.



Etiology :

1. Cervical rib which may be complete or incomplete. This bony structure extends from the 7th cervical vertebra to the first rib.

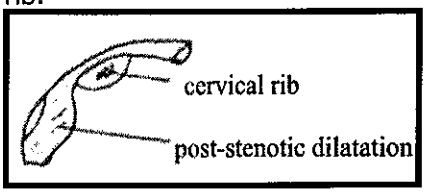
2. A fibrous band extending from an incomplete cervical rib and ending at the first rib.

3. A tight scalene muscle.

4. Post fixation of the brachial plexus. In this case the lower root of the brachial plexus arises from T2 instead of T1 thus this nerve becomes excessively bent over the first rib.

Complications :

- **Post-stenotic dilatation :** It is an aneurysmal dilatation, which may send a shower of emboli to the index and middle fingers, as they are the direct continuation of the brachial artery.



- Compression of subclavain vein produces subclavain & axillary vein thrombosis and chronic venous insufficiency of upper limb.

Clinical picture :

Incidence :

- More commonly in females. About 20 years.
- Usually unilateral affecting the right arm (dominant arm).

Symptoms :

- **Vascular :** Intermittent claudication.
- **Vasomotor :** Raynaud's phenomenon due to irritation of the sympathetic fibers.
- **Nervous :** Tingling and numbness in the medial aspect of the forearm and the hand.

the lower trunk of Brachial plexus is the main structure to be compressed

Signs :

- **A bony swelling** at the base of the posterior triangle (the bony cervical rib).
- **Hyposthesia and wasting** of **hand** muscles may be noticed.
- **Adson's test :** The patient is asked to extend his head, look to the opposite side and take a deep breath. The examiner then palpates the radial pulse while the arm is pulled down. If the test is positive, the pulse will be weaker. **This test is not very accurate.**

Differential diagnosis :

- Spondylosis.
- Carpal tunnel syndrome.
- Other causes of Raynaud's phenomenon .g. Raynaud disease, system lupus.

Investigations :

- **Plain X-ray to the neck** may detect a bony cervical rib.
- **Nerve conduction study** between the neck and the forearm to detect delay in conduction. This is a useful investigation and can differentiate between thoracic outlet syndrome and carpal tunnel syndrome.
- **Subclavian angiography.**

Treatment :

[A] No Symptoms : No treatment.
[B] Physiotherapy : to strengthen the shoulder muscles
[C] Surgery : The following operations:
(a) Excision of a bony cervical rib. The rib should be excised with its periosteum.
(b) Incision of the scalenus anterior muscle (scalenotomy).
(c) Excision of the first rib to relieve the compression inferiorly (very efficient).

N.B. In many persons a cervical rib is detected by radiography, yet it is asymptomatic. Also in many patients who have the clinical picture of thoracic outlet syndrome, radiography does not reveal a cervical rib.

Radical block neck dissection

Definition : Removal of all lymph nodes on one side of the neck, in one mass (en block).

Indications :

- Operable primary malignancy of the head or neck, with palpable malignant cervical lymph node ***(The main indication)***
- Prophylactic radical block neck dissection i.e. where cervical nodes are impalpable e.g. melanoma and carcinoma of the tongue.

Technical considerations :

- **Incisions:** the operation can be done through different incisions; the goblet incision is a popular one

Structure that are removed in radical block neck dissection :

(A) All lymph nodes on one side of the neck
(B) Other structures are also removed because of their close proximity to the cervical nodes :

- Sternomastoid muscle
- Carotid sheath
- Internal jugular vein
- Submandibular salivary gland
- Lower part of the parotid gland

In bilateral block neck dissection, preservation of one jugular vein on the less affected side is necessary

Variations of radical block neck dissection:

- **Suprahyoid block dissection:** it is restricted to the lymph nodes above the level of the hyoid bone *It is indicated in carcinoma of the lower lip & in early cases of carcinoma of tip of tongue & floor of the mouth*

- **Modified radical block dissection:** similar to radical block neck dissection but with preservation of the internal jugular vein, sternomastoid and spinal accessory nerve. *It is indicated in thyroid carcinoma*

Pharyngeal diverticulum (pharyngeal pouch)

A protrusion of mucous membrane through the Killian's dehiscence (pulsion diverticulum) due to raised intrapharyngeal pressure, from failure of relaxation of the cricopharyngeus (achalasia) during contraction of the pharyngeal muscles above it, in the act of swallowing. Once formed, the protrusion fills with food at every meal, and progressively, increases in size displacing the esophageal opening. The pouch descends towards the mediastinum, but often turns outwards, usually to the left to appear in the posterior triangle of the neck.

Clinical features :

- The patient is usually an elderly, and the condition is twice as common in men as in women.
- It may give rise to symptoms identical to those of a foreign body in the throat.
- In long-standing cases there is history of dysphagia, recently aggravated and associated with regurgitation of undigested food after meals, eructation of gas and a gurgling noise in the neck.
- Sometimes, there is an irritable cough from overflow of food into the larynx.
- In about one-third of cases, the pouch is large enough to form a visible swelling in the neck, which enlarges when the patient drinks and can be emptied by pressure.
- In advanced cases, progressive loss of weight occurs and may lead to cachexia.

Diagnosis :

- Barium swallow may show a bulge in the posterior wall of the pharynx in early cases, but in late cases there is a flask shaped pouch sometimes with a fluid level.
- Esophagoscopy is not recommended as it may perforate the fundus of the pouch.
- Manometric studies may be helpful.

Treatment :

When the pouch is of a large size, surgery is recommended. The diverticulum is excised, its neck closed in two layers and a cricopharyngeal myotomy is performed.

Post-Cricoid Carcinoma

Incidence:

- More common in females (around age of 40 years). - May occur on top of Plummer Vinson's Syndrome.

Clinical Picture:

1. Early symptoms: Pain in the throat that is referred to:

- The side of the neck. - Ear (stimulation of Arnold nerve).

2. Foul smelling of breath and loss of weight.

3. Dysphagia is late.

4. Examination:

- Forward bulging of thyroid cartilage and trachea.
- The alae of thyroid cartilage may spread outwards.
- Loss of laryngeal click.

Investigations:

1. Barium swallow. 2. Direct pharyngoscopy and biopsy.

Treatment:

[A] Operable and fit patients:

- Total laryngopharyngectomy and block dissection of lymph nodes.
- Replacement of pharynx by:

1. The stomach is mobilized and pulled up to the neck and anastomosed with the pharyngeal stump.
2. Pectoralis major myocutaneous flap.

- The patient will be left by a tracheostomy.

[B] Inoperable and unfit patients: Treated by external irradiation.

Breast Diseases

The Breast

Congenital Anomalies

Congenital anomalies of the breast may involve the nipple or the breast itself.

Anomalies of the nipple :

1. Athelia : Absence of the nipple is very rare and is usually associated with absence of the breast (Amazia).

2. Polythelia : Supernumerary nipples occur anywhere along either or both the mammary ridges (mammary lines) which extend from the axillae to the groins. An accessory nipple may be mistaken for a mole or a wart.

Anomalies of the breast :

1. Amazia : Absence of the breast is usually unilateral and is often associated with absence of the sternal portion of the pectoralis major.

2. Polymazia : Supernumerary breasts are due to persistence of extramammary portions of the mammary ridges. They may occur in the axilla (accessory breast), groin, or even the thigh. They may function during lactation.

3. Small breast (Micromazia) : Treatment : Augmentation mammoplasty.

4. Diffuse Hypertrophy : Treatment : reduction mammoplasty.

5. Infantile gynecomastia : Diffuse enlargement of the breast, which may be unilateral or bilateral. It is caused by the effect of circulating maternal sex hormones. The condition is usually reversible within 6 months and therefore requires no treatment.

Inflammations of the breast (Mastitis)

Acute lactational mastitis & breast abscess

Incidence : acute mastitis is most common during lactation.

Etiology :

- **Organism :**

Staphylococcus aureus : It induces clotting of milk in the ducts producing obstruction and stasis.

- **Route of entry :**

1. Organisms from the mouth of the suckling infant gain access through nipple cracks and openings of lactiferous ducts.
2. Much less common is the blood born infection.

- **Predisposing factors are :**

1. Milk engorgement caused by inspissated milk and epithelial debris that block the ducts.
2. Abrasions to the nipples.
3. Poor hygiene.

Pathology :

- Infection is at first diffuse.
- The disease usually starts by milk engorgement, which if not properly treated will lead to acute mastitis.
- Staphylococci tend to produce necrosis and pus formation causing a **multilocular abscess.**

Clinical Features :

[A] Stage of milk engorgement :

1. **Pain :** Dull aching.
2. **Fever :** Persistent pyrexia.
3. **Examination reveals :** Enlargement and induration of the breast but there are no physical signs of inflammation.

[B] Stage of acute mastitis :

1. **Pain :** Gets worse.
2. **Fever :** Continuous higher pyrexia.
3. **Examination reveals :** Diffuse swelling, redness, induration and tenderness.

[C] Stage of acute abscess : Suppuration is diagnosed if :

1. **Pain :** Throbbing.
2. **Fever :** Hectic.
3. **Examination reveals :**
   - Physical signs get localized in the breast.
   - Pitting edema is elicited in the area of the skin overlying the abscess.
   - Persistence of the local signs of inflammation for more than 5 days or severe systemic upset for more than 2 days after full antibiotic treatment.

Breast abscess is one of the sites where the surgeon should not wait for fluctuation to diagnose it.

[D] Stage of chronic breast abscess.

Differential Diagnosis : The most serious is acute mastitis carcinomatosa.

Investigations : Total leucocytic count will show leucocytosis.

Treatment :

[A] Before the development of an abscess :

- The condition can be medically treated.

1. **An antibiotic** against staphylococci e.g. flucloxacillin, cephalosporin.
2. **Support of the breast** helps to lessen pain.
3. **Local heat.**
4. **The advisability of weaning is controversial, but a reasonable approach is to :**
   - Advise cessation of breast-feeding if the baby has been nursed for more than 9 months. The agents in common use are bromocriptine (Parlodel) 2.5 mg twice daily or stillbesterol 10 mg three times / day.
   - If the baby is younger, the patient is asked to use the healthy breast for feeding and to regularly empty the inflamed breast by squeezing and by a breast pump. After resolution of infection the baby can be refed by both sides.

[B] Abscess formation is treated by incision and drainage :

- **Anesthesia :** general.
- **Incision :** A radial or more cosmetic circumferential over the most tender area to release the pus, which is sampled for culture and sensitivity.
- The surgeon's finger **breaks down the septa** between the loculi to form a single cavity.
- **A drain** is brought out through the wound or at the most dependant part of the breast through a **counter incision.**
- **Antibiotic** therapy may be continued for a few days.

N.B: some surgeons advise in selected cases of breast abscess repeated aspiration guided by ultrasonography.

Non-lactational mastitis

- The commonest type of non-lactational mastitis is that which complicates mammary duct ectasia.
- Inflammation produces a painful tender periareolar swelling.
- Anaerobes are commonly responsible for mastitis. Therefore, it is customary to use metronidazole (400mg 4 times per day) with flucloxacillin (250 mg 4 times per day) for 5 days.
- If an abscess forms, it needs surgical drainage.
- Mammilary fistula is a possible complication that requires surgical excision.

Pubertal Mastitis

- This condition affects adolescent boys leading to pain and swelling of the breast, which becomes indurated and slightly tender, but suppuration never occurs.
- The tenderness usually subsides in two years, but may persist for a prolonged period.

Chronic Inflammatory Conditions of the Breast

1. Mammary duct ectasia (plasma cell mastitis).

2. Chronic breast abscess :

- **Etiology :** Acute abscess, which was treated by, prolonged use of antibiotics or was drained by a small incision.
- **Clinical picture :** See carcinoma.
- **Treatment :** Treatment is excision not only simple incision.

3. Tuberculosis of the breast :

- **Clinical picture :** see carcinoma.
- **Treatment :** Is by antituberculous drugs. Mastectomy is indicated in patients with resistant infection.

Mammary Duct Ectasia

- The disease is characterized by dilatation of the major ducts, which fill with creamy secretion.
- The etiology is unknown.

- Duct ectasia may be asymptomatic or may present by one of the following :
  1. **Nipple discharge :** blood stained, serous, creamy white or yellow.
  2. **Retraction of the nipple :** due to shortening of the ducts.
  3. **Acute inflammation**
  4. **Recurrent and chronic inflammation :** see breast carcinoma.

- **Treatment :**
  Troublesome cases of duct ectasia are treated surgically by **excision of the major ducts** through a circumareolar incision.

Fibrocystic disease of the breast

Incidence :

- **This is the most frequent disorder of the breast.**
- The upper outer quadrant of the breast is the commonest site of affection.

Other names :

Mammary dysplasia, **ANDI** (aberrations of normal development and involution), fibroadenosis.

Etiology :

- The exact cause is not known, but the prevalence of the disorder in women aged 30-50 years and its rarity after the menopause suggests that it is related to ovarian activity.

Pathology :

Mixture of the following with a variable proportion :

1. Adenosis : Glandular hyperplasia- causing multiplication of acini.

2. Epitheliosis : Solid epithelial hyperplasia within small ducts. In rare cases there is **"Atypical epithelial hyperplasia"** which is accompanied by higher possibility of developing breast cancer.

3. Fibrosis : When fibrosis is extensive it clinically resembles schirrhous carcinoma, and is called **"sclerosing adenosis".**

4. Cyst formation :

- **Structure :**

(a) Lined with epithelium & are filled with clear yellow or sometimes brownish fluid.
(b) Cyst may be large, distended with clear fluid and the cyst is now called (Blue doomed cyst of Bloodgood)

- **Etiology :**

(a) Abnormal involution of the supporting connective tissue in the lobules allows the acini to dilate.
(b) Obstruction of the ducts by papillomatosis and by the surrounding fibrosis.

Clinical Features :

1. May be asymptomatic.

2. Accidentally felt lump. This may be caused by a prominent cyst, aggregation of small cysts, or by the sclerosing adenosis.

3. The most frequent complaint is of multiple, sometimes painful, small lumps that may be unilateral or bilateral **(painful nodularity).** The lady usually observes this in relation to her cycles; on examination they are felt by **fingertips** not by flat of the hand.

4. Mastalgia (breast pain) : Usually cyclical. The pain typically occurs few days premenstrually and is accompanied by enlargement and increased nodularity of the breasts.

5. Nipple discharge : Is usually clear or yellow, but sometimes brown or green.

Differential Diagnosis :

1. Other causes of breast mass.
2. Other causes breast cysts.
3. Other causes of mastalgia.

Investigations :

- Investigations are usually not required, but the following are indicated to rule out breast cancer in suspected cases :

1. Ultrasound and mammography : show the cysts.
2. A cyst is fully aspirated. It is considered benign if :
(a) The fluid is not blood stained.
(b) The mass disappears completely after aspiration.
(c) Does not recur within two weeks.
(d) Cytology of the fluid may be performed to exclude malignancy, but the results are not conclusive.
3. A solid mass is aspirated for cytology.
4. Open biopsy : is indicated if cytology is not available, or if its result is not conclusive.

Treatment :

Exclusion of malignancy and reassurance of the patient are the most important.

- **No symptoms :** No treatment.
- **Cysts :** Are treated by aspiration. A recurring cyst is excised for biopsy.
- **Cyclic mastalgia :**

1. In mild cases : reassurance and wearing (night and day) a brassiere is usually enough.
2. Giving up caffeine consumption (coffee, tea and chocolate) may be useful.
3. Analgesics : NSAID's
4. Prolactin inhibitor : as bromocriptine 2.5 mg b.d. gives good results in many patients.
5. Danazol : which is a synthetic androgen, is effective in controlling cyclic pain.

- **N.B. Cases with atypical epithelial hyperplasia need regular medical follow up.**

Cysts of the breast

Classification :

[A] Acinar Cysts : (From the duct system)

1. Retention cysts : See fibroadenosis.
- Large cysts distended with clear fluid may have a blue green color (blue domed cyst of Bloodgood).
- It represents more than 75% of breast cysts.

2. Galactocele (milk cyst) :
- Develops during or shortly after lactation from obstruction to one of the principal ducts.
- The cyst forms a solitary painless swelling situated close to the nipple; a little milky fluid may be expressed from the nipple by pressure on the cyst.

3. Intracystic papilliferous carcinoma.

[B] Inter-acinar cysts : (in the stroma)

- **Cysts arising in the stroma are rare and include :**

1. Dermoid cyst.
2. Lymph cyst.
3. Blood cyst : arise either as traumatic hematoma or due to bleeding into a cyst.
4. Hydatid cyst.
5. Sebaceous cysts.

Treatment:

1. Aspiration : Mention criteria of benign and suspicious cyst.
2. Excision : Retention cysts, which recur after aspiration, and all other cysts should be excised.

Nipple discharge

Etiology :

[A] Physiological :

1. Milk production with lactation.
2. Serous discharge during pregnancy.

[B] Pathological :

Cause	Nature of discharge
Duct ectasia (the commonest)	Serous, creamy white, yellow, blood stained, brown-green or black
Fibrocystic disease	Clear, yellow, brown or green
Duct papilloma	Blood or blood stained discharge from the duct
Duct carcinoma	Rare cause of blood or blood stained discharge from one duct
Contraceptive pills	Serous or milky discharge from multiple ducts
Hyperprolactinemia	Milky discharge (galactorrhea) from multiple ducts

Diagnosis : History and examination should provide the following information :

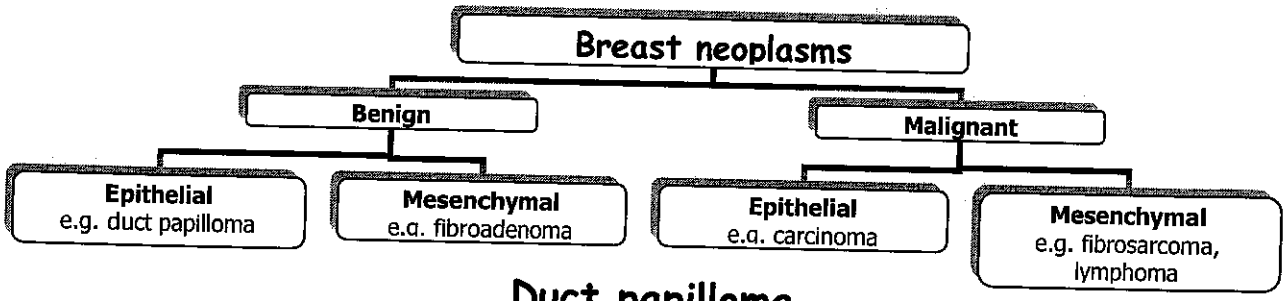
1. Nature of discharge.
2. Association with a mass.
3. Unilateral or bilateral.
4. Single duct or multiple duct discharge (Differential pressure test).
5. The use of contraceptive pills.

Investigations :

1. Test for occult blood : in the discharge if it is not apparent.
2. Cytology : of the discharge for exfoliated cancer cells.
3. Soft tissue mammography.
4. Duct galactography : in case of single duct discharge.
5. Serum prolactin estimation : in suspected cases of galactorrhea.
6. Ultrasound : to detect solid or cystic swelling.
7. Biopsy : if mass was detected with mammography or ultrasound.

Treatment :

1. A palpable mass should be **excised** for histology and treated accordingly.
2. A single duct bloody discharge calls for excision of this duct **(microdochectomy).** A needle with a blunt tip is introduced in to the affected duct & acts as a guide for the surgeon. This duct, with a rim of the surrounding tissues is excised and is sent for histological examination.
3. Discharge from multiple ducts with no palpable or mammographic mass is treated **conservatively by observation.** Rarely the discharge is persistent and troublesome where it is surgically treated by **excision of the major ducts.**



Duct papilloma

- A duct papilloma is usually situated in one of the main ducts near the nipple in a young woman.

Clinically : 1. Bloody or blood stained discharge (the commonest).
2. Retention cyst : Behind the mass.
3. Palpable mass : Pressure on the mass may produce discharge.

Ductography : Shows filling defect.
Treatment : Is by excision of the affected duct (microdochectomy).

Fibroadenoma

- Fibroadenoma is the commonest breast mass of young women. The usual age is between 15-30 years.

Pathology :

A fibroadenoma is a benign mixed neoplasm of the breast, which affects both the fibrous and the glandular tissues, but the fibrous element predominates.

Grossly :

- The tumor may be solitary or multiple, firm, with smooth surface that may be lobulated in big lesions. It is well circumscribed and is never attached to surrounding tissue.
- The cut surface reveals lobules of whirled white fibrous tissue, which bulges out of its capsule.

Histologically : There are two types :

- **Pericanalicular (hard) fibroadenomata :** Are the usual form. These tumors are formed mainly from fibrous tissue that surrounds a few small tubular glands. They tend to be small.
- **Intracanalicular (soft) fibroadenomata :** Contain more glands. They are usually larger and softer than the pericanalicular type.

Clinical Features :

- **Age :** Hard fibroadenoma occurs commonly in young women 20-30 years of age while soft fibroadenoma occurs between the ages of 30-50.
- **Painless lump** accidentally discovered usually small (few centimeters), not tender, well circumscribed with smooth surface that may be lobulated with high mobility within the breast **" breast Mouse ".**

Items	Hard fibroadenoma	Soft fibroadenoma
Frequency	Commoner	Less common
Age	20-30 years	30-50 years
Size	Small	Large
Shape	Rounded	Lobulated
Consistency	Firm	Softer
Mobility	Free (breast mouse)	Restricted
Complications	Never turn malignant	Liable to turn to sarcoma

Investigations : Soft tissue mammography reveals well-circumscribed lesion.

Treatment : Treatment is by **excision** and histological confirmation of the diagnosis.

Cystosarcoma Phylloides

Pathology : Highly cellular type of fibroadenoma that tends to grow rapidly.

The name : It was so named by Brodie who used the term phylloides because the cut surface resembles a leaf.

- The term Cystosarcoma however is a **misnomer** as many are not cystic and it is rarely malignant.
- It is therefore better termed **Phylloides tumor.**

A big tumor may cause skin ulceration. The tumor is not attached to the skin.

Treatment : Is by wide local excision to prevent recurrence. If the tumor is occupying the whole breast, simple mastectomy is indicated.

Carcinoma of the breast

Introduction :

- The breast is the most common site of cancer in women.
- In the USA one in every nine women is expected to develop cancer in lifetime.
- In Egypt it occurs at a younger age than in western countries and it accounts for about 35 % of the total malignancies among Egyptian females representing the commonest malignant neoplasm in females.

Incidence :

- **Race :** Black and Asian women are less affected.
- **Age :** In high-risk population, the risk generally increases progressively with age. In low risk population, the risk increases and reaches a plateau at the age of 50.

Etiology : The exact etiology of breast cancer is not known. Genetic, endocrinal or environmental factors may have role.

[A] Genetic factors :

- It has been proved that in 5-10% of all cases of breast cancer, there is an autosomal inheritance of a mutant gene. Two genes BRCAI (on chromosome ***17***) & BRCAII (on chromosome ***13***) are incriminated. These cancers usually occur at a younger age & are multifocal & bilateral.
- Presence of breast cancer in the mother or sister increases the risk by 2-3 times while presence of the disease in both the mother & sister increases the risk by 14 times.

[B] Endocrinal factors :

1. **Cancer in the other breast.**
2. **Atypical epithelial hyperplasia.**
3. **Menstrual history :** Early menarche (under 12 years), late menopause (after 50 years).
4. **Obesity & high intake of saturated fatty acids :** Steroid hormones are converted to estradiol in fatty tissues.
5. **Pregnancy :** late first pregnancy (after age of 30), nullipara.
6. **The relation to oral contraception** is not exactly known.

[C] Precancerous lesions :

1. **Moderate or marked epithelial hyperplasia & duct Papilloma :** increase the risk by 1.5–2 times.
2. **Atypical epithelial hyperplasia :** increases the risk by 2–5 times.
3. **Lobular or ductal carcinoma in situ :** increases the risk by 5–10 times.

Site :

- **Inner, lower (6%) :** Is the rarest & worst → spread to subdiaphragmatic lymphatics.
- **Upper, outer (60%) :** The commonest → largest volume of breast tissue.
- **Central part (12%) :** The 2nd common → spread to subareolar area from neighboring tissue.
- **Lower, outer (12%).**
- **Upper, inner (10%).**

Pathology : There are the following pathological types :

[A] Carcinoma of the ducts :

1. Non-infiltrating (ductal carcinoma in situ) : 6%	2. Infiltrating ductal carcinoma :
(a) Comedocarcinoma. (b) Non-comedocarcinoma. (Papillary or solid).	(a) Schirrhous carcinoma 70-75%. (b) Encephaloid carcinoma 10%. (c) Mucinous carcinoma 3%.

[B] Carcinoma of the lobules :

1. Non-infiltrating lobular carcinoma (in situ) 0.7%.
2. Infiltrating lobular carcinoma 7.5%.

[C] Paget's disease of the nipple.

[A] Carcinoma of the ducts

1. Non-infiltrating (ductal carcinoma in situ) (6%)

- **Non-infiltrating comedocarcinoma :** Central necrosis of cancer cells leads to the extrusion of a sebaceous like material from the cut surface of the tumor.
- **Non-infiltrating papillary duct carcinoma :** May cause bleeding from the nipple.

2. Infiltrating ductal carcinoma

- **Schirrhous carcinoma : (70-75%) (NOS = Not Otherwise Specified)**

The tumor : Is hard. The cut surface is concave, rough, gritty and pale gray.
Microscopically : All degrees of differentiation from anaplasia to well differentiated tumors with attempts at adenoid formation may occur.
Spread : Early lymphatic spread.

- **Encephaloid carcinoma : (10%)**

The tumor : Is Large, soft and brain like.
Microscopically : It contains less fibrous tissue than **NOS**. The tumor shows lymphocytic infiltration.
Spread : To lymph nodes occur later than **NOS**. The prognosis is better than **NOS**.

- **Mucinous carcinoma : (3%)**

The tumor : Has a gelatinous consistency and is usually bulky.
Microscopically : Mucin is produced by the tumor cells and is present either intra or extra cellular.

- **Inflammatory carcinoma (mastitis carcinomatosa) :**

The tumor : Is a rare type, but is the most malignant. It is a fulminant form that resembles mastitis.

[B] Carcinoma of the lobules

- **Non-Infiltrating lobular carcinoma : (0.7 %)**

Frequently multicentric.

- **Infiltrating lobular carcinoma : (7.5 %)**

1. Grossly and microscopically : The tumor is indistinguishable from scirrhous carcinoma and the diagnosis can be certain only when the preinvasive lobular nodule of a tumor can be identified histologically.
2. Bilateral in 25 % of cases.

[C] Paget's disease of the nipple (1%)

1. Nature : An intraduct carcinoma which begins in the epithelium of a main duct and spreads to the skin of the nipple and down into the breast substance.
2. When the nipple epithelium is infiltrated, it produces nipple erosion. In early stages a mass may not be palpable, a mass may only appear after 2 years from the start of the disease.
3. Histologically : there are :
(a) Paget's cells : Clear vacuolated cells, with small dark staining nuclei, which occur alone or in clusters in the deep layers of the epidermis.
(b) Hyperplasia of all layers of the epidermis.
(c) Round cell infiltration of the dermis.
N.B:James Paget(1809-1892) is a pathologist-university of cambridge.

Hormone receptors :

- About 60% of breast cancers have receptors for estrogen & are termed ER-positive.
- These tumors are hormone dependant & respond more to hormonal treatment.
- Tumors may also have progesterone receptors.
- Recently: tumors are stained for ***c-erb 52*** (growth factor receptor). if proved to be positive,monoclonal antibodies against these receptors could be used.

N.B: *li-fraumini syndrome:* Mutation in tumor suppressor gene p53 leading to: Malignancies in breast, ovary, colon and lymphomas.

Spread :

- Previously it was thought that breast cancer spreads locally at first, then by lymphatics and lastly by the blood stream.
- This view is no longer accepted and is now well realized that carcinoma of the breast may spread by the blood stream very early producing **micro-metastases** in distant organs.

[A] Local spread :

- Can occur through the breast substance, overlying skin- underlying pectoralis major and serratus anterior muscles and the chest wall.

[B] Lymphatic spread :

- **By embolism and permeation** is mostly to the axillary nodes, next common is the internal mammary chain.
- **Involvement of the supraclavicular node** is considered an advanced disease.

1. Axillary lymph nodes are classified into 3 levels :

- **Level I** : Below the pectoralis minor muscle.
- **Level II** : Behind the pectoralis minor muscle.
- **Level III** : Above the pectoralis minor muscle.

2. Mammary lymph nodes lying along the internal mammary vessels in the first three intercostal spaces. They receive part of the lymph from the medial half of the breast.

3. A few lymphatics pierce the pectoralis major muscle to drain into the inter-pectoral L.N. **(lymph nodes of Rotter)** and pass to the posterior intercostal lymph nodes.

4. Lymphatics from the lower inner quadrant : may pierce the rectus sheath to reach the peritoneal lymphatic plexus.

5. Supraclavicular lymph nodes : may be affected by retrograde lymphatic spread.

6. Obstruction of the dermal lymphatics by malignant cells produces breast skin edema that is marked in the dependant part, i.e. the lower half of the breast. The edematous skin is pulled at the sites of hair follicles, sebaceous glands and sweat glands giving the appearance of an orange peel, hence the French name **Peau d' Orange.**

[C] Blood stream spread :

- Produces metastasis in the lungs, bones, brain and liver.

N.B: part of the venous drainage of the breast goes to intercostal veins which drain into the azygos system and communicates with valveless vertebral venous plexus. This explains the tendency of breast cancer deposits to affect the axial skeleton

Staging :

[A] International TNM staging :

T = Tumor

Tis : Carcinoma in situ. Paget's disease with no palpable tumor.
T0 : No evidence of primary tumor.
T1 : 2 cm diameter or less.
T2 : 2-5 cm diameter.
T3 : Tumor larger than 5 cm.
T4 : Any size with direct extension to chest wall or to skin.

N = Nodes

N0 : No palpable axillary nodes.
N1 : Mobile palpable homolateral axillary nodes.
N2 : Fixed homolateral axillary nodes.
N3 : Palpable homolateral supraclavicular nodes. Edema of the arm.

M = Metastases

M0 : No evidence of distant metastases.
M1 : Distant metastases.

[B] Staging of the UICC (union international center cancer) is now internationally approved. It is based on the TNM staging:

Stage UICC		description	category	5 year survival (%)
I		T1,N0,M0	Early breast cancer	93
II	IIA	T2,N1,M0	Early breast cancer	72
	IIB	T3,N0,M0		
III	IIIA	T1-3,N0-2,M0	Locally advanced Breast cancer	41
	IIIB	T4,any N,M0		
IV		Any T,any N,M1	metastatic	18

[C] Manchester classification :

Stage I :
- A mobile mass in the breast.

Stage II :
- A mobile mass in the breast with or without skin tethering.
- Palpable mobile homolateral axillary nodes.

Stage III :
- Peau d' orange larger than the tumor but still limited to the breast.
- Tumor fixed to pectoral muscle but not to the chest wall.
- Homolateral axillary lymph nodes matted together or fixed to chest wall.
- Homolateral supraclavicular lymph nodes.
- Edema of the arm.

Stage IV :
- Skin involvement wide of the breast (cancer en cuirasse).
- Fixation to the chest wall.
- Distant metastases.
- Involvement of the opposite breast or axilla.

Clinical Features :

I- Symptoms :

1. The patient accidentally notices **a painless lump in the breast.** An accidental trauma to the breast may attract the attention of the patient to presence of the lump.
2. Much less frequently the disease is discovered because of **mild breast pricking pain, nipple retraction** or **blood stained nipple discharge.**
3. Picture of metastasis (lymph node or blood).
4. The disease may be discovered by **routine screening mammography.**

II- Signs : Some of the following signs may be detected :

[A] Breast :

1. Asymmetry. 2. Enlargement.
3. Skin dimpling. 4. Skin puckering.

- **Dimpling and puckering of the skin are evident when the patient is sitting and elevates her arms.**

5. Peau d'orange (orange peel). 6. Skin nodules. 7. Skin ulceration.

- **Peau d'orange, skin nodules, and ulceration indicate a locally advanced disease.**

[B] Mass :

1. Hard. 2. Irregular. 3. Ill-defined.
4. Restricted mobility within the breast substance.
5. Fixation to the skin, underlying muscles, or chest wall, if present, is diagnostic of carcinoma.

[C] Nipple :

1. Recent retraction. 2. Change of direction.

[D] Axillary and supraclavicular nodes :

- **Number and mobility of palpable nodes are assessed.**

[E] Distant metastases :

1. Chest examination. 2. Hepatomegaly. 3. Ascites.
4. Pelvic examination for hard deposits or Krukenberg tumor.

Skin manifestations of breast cancer

1. Dimpling and puckering of the skin : Overlying the tumor. This is the earliest manifestation of superficial tumors. It is due to infiltration of Cooper's ligaments by the tumor, pulling the skin toward the tumor.(Cooper's ligament : Are conical fibrous tissue normally connecting the skin to the pectoral fascia).
2. Fixation.
3. Fungation.
4. Ulceration : Ulcer is always indurated.
5. Skin nodules : Near or far from the tumor, even around the umbilicus. They are due to lymphatic spread.
6. Peau d' orange: non-pitting edema due to obliteration of the skin lymphatics by malignant permeation & surrounding fibrosis. The pits giving skin the orange appearance are due to hair follicles, sweat glands & sebaceous glands anchored to the skin.
7. Cancer en cuirasse : In very late cases, due to malignant infiltration and fibrosis of the skin as well as lymphatic obstruction, the skin is very hard, very thick, and non-mobile like a shield.
8. Nipple : Is retracted.
9. Paget's disease of the nipple.

III- Special clinical forms :

1. Paget's disease of the nipple : (1 % of breast cancers)

- The first symptom is often an abnormal pricking sensation of the nipple, with superficial erosion.
- A tumor mass may not be palpable.
- The lesion is commonly mistaken for eczema.
- The diagnosis is established by biopsy of the erosion

	Paget's disease	Eczema
1	Unilateral	Commonly bilateral
2	Usually occurs at menopause	Commonly occurs at lactation
3	No itching	Itching
4	No vesicles-not oozing (dry)	Vesicles-oozing
5	Nipple is eroded	Intact nipple
6	Well-defined margin	Ill defined margin
7	A breast lump may be felt	No lump
8	No response to eczema treatment	Responds to treatment
9	Starts in the nipple	Starts in the areola

2. Inflammatory carcinoma :

- This is a rare aggressive form of breast cancer.
- Usually occurs during pregnancy or lactation.
- There is a rapidly growing, sometimes painful, breast swelling.
- The overlying skin becomes red, edematous and warm. Often there is no distinct mass, since the tumor infiltrates the breast diffusely.
- The picture clinically resembles acute mastitis.
- **Prognosis :** is poor as it is usually advanced at the time of diagnosis.

Inflammatory carcinoma	Acute bacterial mastitis
Gradual onset with no or low grade fever	Acute onset with high fever
Progress is slower	Rapid progress
Involves more than one third of the breast	One breast sector is affected
Skin is dusky red	Skin is rosy or bright red
Mildly tender or non-tender lesion	Markedly tender lesion
Non-tender axillary nodes	Tender axillary nodes
No response to antibiotics in one week is an indication for biopsy	The lesion is either cured by antibiotics or forms an abscess

3. Carcinoma in situ:

	Duct carcinoma in situ (DCIS)	Lobular carcinoma in situ (LCIS)
Frequency	More common	Less common
Bilaterality and multicentricity	Rare	common
Microcalcifications	Present	absent
Early detection	Possible	Less likely
Potential for invasive cancer	30-50%	It is a marker of increased risk of malignancy in the same or other breast
Treatment	As invasive cancer	Strict follow up.

Differential Diagnosis :

I- Case presenting by a lump (mass):

Items	1. Carcinoma	2. Solitary cyst	3. Fibrocystic disease	4. Fibroadenoma
Age	Usually > 35 yrs	35-55 years	20-55 years	15-30 years
Pain	Painless	Occasionally painful	Occasionally painful	Painless
Surface	Irregular	Smooth surface	Indistinct surface	Smooth may be lobulated
Consistency	Stony hard	Fluctuation is difficult to elicit, so it feels soft to hard	Firm, ill defined areas of thickening	Firm, highly mobile
Lymph nodes	Probably axillary node enlargement	Free axilla	Free axilla	Free axilla

The above lesions constitute 95 % of breast lumps.

Other less common causes are :

5. Traumatic lesions : hematoma and fat necrosis.

Breast hematoma	Traumatic fat necrosis
• If there is no external bruising, a deeply seated old hematoma may form a hard mass that greatly resembles a carcinoma.	• Blunt breast trauma may cause death of some of the fat cells. • The liberated fatty acids combine with calcium to form calcium soaps. • The result is one of two forms : 1. A cyst that contains thick oily fluid is formed. 2. Less frequently a hard mass, which resembles a carcinoma, is formed.

6. Inflammatory lesions : Chronic abscess, mammary duct ectasia & T.B.

Chronic breast abscess	T.B.	Mammary duct ectasia
• This is the result of improper treatment of acute abscess. • There may be (with the mass) nipple retraction and skin puckering. • However it is more painful than carcinoma and is accompanied by low-grade pyrexia.	• Presents as : 1. Multiple cold abscesses & sinuses. **OR** 2. Multiple nodules in the breast substance.	• The affected area is hard & may be associated with skin dimpling & nipple retraction simulating carcinoma. • It is called **plasma cell mastitis** due to predominance of plasma cells.

7. Other cysts : e.g. Galactocele.
8. Other tumors : Duct papilloma accumulating blood behind it and sarcoma (very rare).
9. Swellings arising from the chest wall : Tuberculosis or tumor of a rib or lipoma.

II- Paget's disease : Should be differentiated from eczema of the nipple.

III- Inflammatory carcinoma : Should be differentiated from an acute breast abscess.

IV- Nipple retraction : Two types of nipple retraction should be identified :

- **Longstanding type** dates back to the puberty is an innocent finding. It poses difficulty only during lactation. It predisposes to mastitis and resolves spontaneously after lactation.
- **Recent nipple retraction** in womanhood is considered seriously. One cause is carcinoma. Other causes are *mammary duct ectasia, TB* and *chronic breast abscess.*

Investigations :

[A] Laboratory: CEA and Cancer antigen CA 15-3.it is prognostic rather than diagnostic.

[B] Mammography :

- Is soft tissue radiology of the breast. In expert hands it is 95% accurate in diagnosing breast cancer.
- It is useful in detecting multifocal lesions in the same or other side.
- A cancer appears as a dense opacity containing microcalcifications and has an indefinite outline.
- **N.B1:** only 20% of microcalcifications are malignant.

N.B2: xero-radiography: as mammography but more accurate.

[C] Ultrasonography:

- A speculated hypo-echoic mass more deep than wide suggests a malignant lesion.
- It differentiates between chronic abscess & carcinoma.
- It differentiates between solid & cystic lesions.
- Malignant lesion receives blood flow from all around with turbulent speed.
- It is particularly useful in young women inwhom mammography is not helpful.

[D] Magnetic resonance imaging (MRI): it is indicated with contrast in certain situations ***e.g.***

- Postoperative scarring to differentiate between fibrosis and local recurrence of malignancy.
- After neoadjuvant therapy to monitor response.
- In the presence of breast implants

E] Biopsy:

1. **Excision biopsy:** Is the most reliable and provides a big specimen to allow for hormone receptor estimation as well.
2. **Frozen section biopsy :** The biopsy is frozen and slides are prepared from the frozen block. A diagnosis is obtained within 20 minutes. Patient is kept under general anesthesia. If the result is positive, the surgeon proceeds with radical surgery. Consent for mastectomy should be obtained preoperatively.
3. **True-cut biopsy:** done under local anesthesia with a special needle that cuts a core of tumor tissue.
4. **Fine needle aspiration cytology (FNAC) :** It depends on examination of cells to detect criteria of malignancy in them-The aspiration can be done in the outpatient clinic using fine needle. A skilled cytologist is needed.
   - Advantages of FNAC :
     1. Very simple, inexpensive and accurate procedure.
     2. Give a definite diagnosis in 90 % of cases.
5. **Biopsy from impalpable breast masses:** the radiologist can place a wire inside the lesion under mammographic guidance. At operation, the mass is removed with the wire for histological assessment.

[F] Aspiration of a cyst :

- **A breast cyst is considered benign if :**
  1. The fluid is not blood stained.
  2. The cyst disappears completely with aspiration.
  3. Does not recur within 2 weeks.
  4. In case of doubt, the fluid is subjected to cytological examination.

[G] Investigations to detect distant metastasis: routinely done by:

1. Chest x-ray.
2. Ultrasound examination of the pelvis and abdomen
3. Alkaline phosphatase (a high level indicates bone or liver deposits)
4. Further tests (needed in special situations):
   a) Bony pains require x-rays and isotope bone scan.
   b) Suspicion of cerebral secondaries is an indication for CT examination.
   c) PET (positron emission tomography) scan.

Triple assessment

It means comparing the results of

1) **Clinical examination**
2) **Mammography or ultrasonography**
3) **FNAC**

- ***If the three parameters are concordant, the surgeon can rely on the diagnosis.***

Early detection

1. Breast self examination :

All women over age of 20 years should be advised to examine their breasts monthly, one week after the menstrual period. The physician instructs the woman as how to conduct a systematic inspection and palpation.

2. Screening programs :

In some Western countries, high risk women are subjected to regular clinical examination and mammography, every one or three years.

Treatment

I. Early (potentially curable) breast cancer

This is defined as stages I and II in UICC staging.

[A] Surgery:

Different surgical options are available (all of them give almost equal results):

[1] Conservative therapy:

It includes:

1. Local wide excision with a 2cm safety margin. If the lesion is close to skin, part of it may be excised.
2. The sentinel lymph node: is the first node in the axilla to be affected. injection of patent blue violet or a radioactive sulphur colloid near the tumor will allow identification, excision and immediate pathological examination of the sentinel node
   - If the node is positive for metastasis: axillary clearance is done.
   - If the node is negative for metastasis: no further excision of lymph nodes.
3. Postoperative radiotherapy for 4 weeks directed to the breast.

Advantages :

1. If done for the properly indicated patients, the operation provides good results that are equal to radical mastectomy.
2. The breast is preserved minimizing psychological trauma.

Indications :

1. Small tumor ≤ 4cm.
2. Sometimes, large lesions (up to 5 cm) in large breasts.
3. Peripheral lesions.

Contraindications :

1. Pregnancy.
2. Large or central tumors in small breasts (no cosmetic advantages).
3. Multicentric disease as detected by soft tissue mammography.
4. Collagen vascular disease (poor tolerance to radiotherapy).
5. In situ breast cancer more than 20% due to the common incidence of multicentricity.

[2] Modified radical mastectomy (of Patey):

- It involves removal of the following structures in one block:
  - Skin ellipse over the tumor with at least 5 cm safety margin including the areola and nipple.
  - The whole breast including the tumor.
  - The pectoralis minor muscle is either removed or its tendon is cut to open the axilla.
  - All axillary lymph nodes and axillary fat medial to the axillary vein.

NB. Axillary vessels, axillary nerve, nerve to serratus ant. and nerve to latissimus dorsi are spared

After Patey's mastectomy radiotherapy is advised for patients with :
1. Positive axillary lymph nodes. 2. Tumors in the medial half of the breast.

[B] Adjuvant chemotherapy and hormonal treatment:

1. **Hormonal therapy: For all hormone receptor positive cases.**
   It reduces ipsilateral and contralateral breast recurrence by 40%.**tamoxifen** blocks estrogen receptors and **anastrazole** is an aromatase inhibitor which inhibits peripheral conversion of androgen to estrogen.
2. **Chemotherapy: is indicated for:**
   1. Positive axillary nodes.
   2. All patients below 70 years.
   3. Tumors more than 1 cm.
   4. Hormone receptor negative and Her2/neu positive tumors (denote aggressive tumors).
3. **Targeted therapy:** for Her2/neu positive tumors. Monoclonal antibodies are given against Her2/neu receptors **(herceptin)**.

[C] Follow up :

- After treatment, patients are reviewed at regular intervals, usually 3 months for the first 2 years, 4 monthly for the next 3 years, and annually thereafter. **This is required to :**

1. Detect and treat complications of mastectomy :

(A) Psychiatric morbidity : caused by loss of the breast.

(B) Arm edema results from :

- Excision of lymphatics.
- Lymphatic obstruction by radiotherapy.
- Lymphangitis caused by infection.
- Malignant axillary recurrence blocking them.
- Thrombosis of the axillary vein.

How to avoid ?

- Avoidance of radiotherapy to the axilla, which has been surgically evacuated of its nodes, reduces the possibility of lymphatic edema.
- The patient is warned to avoid minor trauma to the ipsilateral hand & should wear gloves when carrying out rough work in order to avoid infection & lymphangitis.

Treatment : (difficult) Arm elevation, massage & elastic or pneumatic arm compression are partially effective.

N.B1: brawny edema: hard but still pitting edema due to infiltration of lymph vessels.
N.B2: elephantiasis surgica: lymphedema secondary to block dissection of lymph nodes.
N.B3: stewart-treves disease: it is lymphangiosarcoma on top of long-standing lymphedema.

2. Detect local recurrence or distant disease.

3. To do annual mammography of the contralateral breast.

4. Ladies who are free of the disease after two years can be considered for reconstruction of breast using either a synthetic implant or a myocutaneous flap (e.g. Transverse Rectus Abdominus Myo-cutaneous flap TRAM). Some surgeons prefer immediate reconstruction for low risk patients.

5. Patients are instructed : Not to get pregnant for at least three years & to use non-hormonal contraception.

II. Treatment of Intermediate disease (locally advanced breast cancer)

- **It includes** tumors larger than 5 cm or fixed axillary or internal mammary nodes.
- Distant metastasis should be excluded.
- **Treatment:** Neoadjuvant (pre-operative) chemotherapy with the aim of down staging of the tumor. Then either conservative surgery or modified radical mastectomy is done according to the response to treatment.

III. Advanced (incurable) breast cancer

- It includes **stages III** and **IV** in UICC staging
- fungation, ulceration & inflammatory carcinoma are included in this category.
- **Endocrine therapy and chemotherapy are the two main lines of treatment.**
- Surgery and radiotherapy are of secondary value.

[A] Endocrine therapy:

Patients who are more likely to respond are :

1) Postmenopausal women.
2) Estrogen receptor positive tumors.
3) Progesterone receptor positive tumors.

The methods in common use are :

1. **Tamoxifen** (Nolvadex tablets): the first line hormone therapy. It is an antiestrogen that blocks estrogen receptors preventing the hormone from activating cancer cells. The dose is 10 mg twice daily. It is ***not*** given for more than 5 years to avoid risk of endometrial cancer or thrombogenecity.
2. **Aromatase inhibitors.**
3. **Raloxifen**:as tamoxifen with less side effects.

[B] Chemotherapy : *It is indicated for the following cases :*

1) Rapidly progressive disease.
2) Premenopausal women.
3) Receptor negative cases.
4) Hormone failure.
5) Liver metastases.

Combinations commonly used include : **C**yclophosphamide, **M**ethotrexate and 5-**F**lurouracil **(CMF).**

N.B: it can be used as a targeted therapy for Her2/neu positive cases.

[C] Radiotherapy: *It is indicated for the following cases:*

1. **Pain** whether due to bone or soft tissue involvement.
2. To control tumor fungation.

[D] Surgery : *It is indicated for the following cases :*

- *Radical mastectomy* may be done for advanced local disease (stage III). It provides good local control.
- *Simple mastectomy* to remove fungating tumor.

[E] Management of specific problems :

1. Pleural effusion : Commonly responds to systemic therapy and chest tube drainage. If not, local instillation of the cytotoxic bleomycin through the tube may be required.

2. Pathological fractures :
Internal fixation is used. The patient is given radiotherapy to the fracture site.

3. Spinal cord compression : requires urgent surgical cord decompression, with stabilization followed by radiotherapy.

4. Cerebral metastases : are treated by a combination of corticosteroids and radiotherapy. Occasionally a solitary brain metastasis is suitable for surgical excision.
The requirements are:
(a) Well-controlled breast primary. (b) A long life expectancy. (c) An accessible area in the brain.

5. Liver metastases : Chemotherapy.

6. Hypercalcemia :

Treatment: Is correction of dehydration by IV fluids + frusemide + prednisolone.

7. Superior vena caval obstruction : Requires urgent radiotherapy.

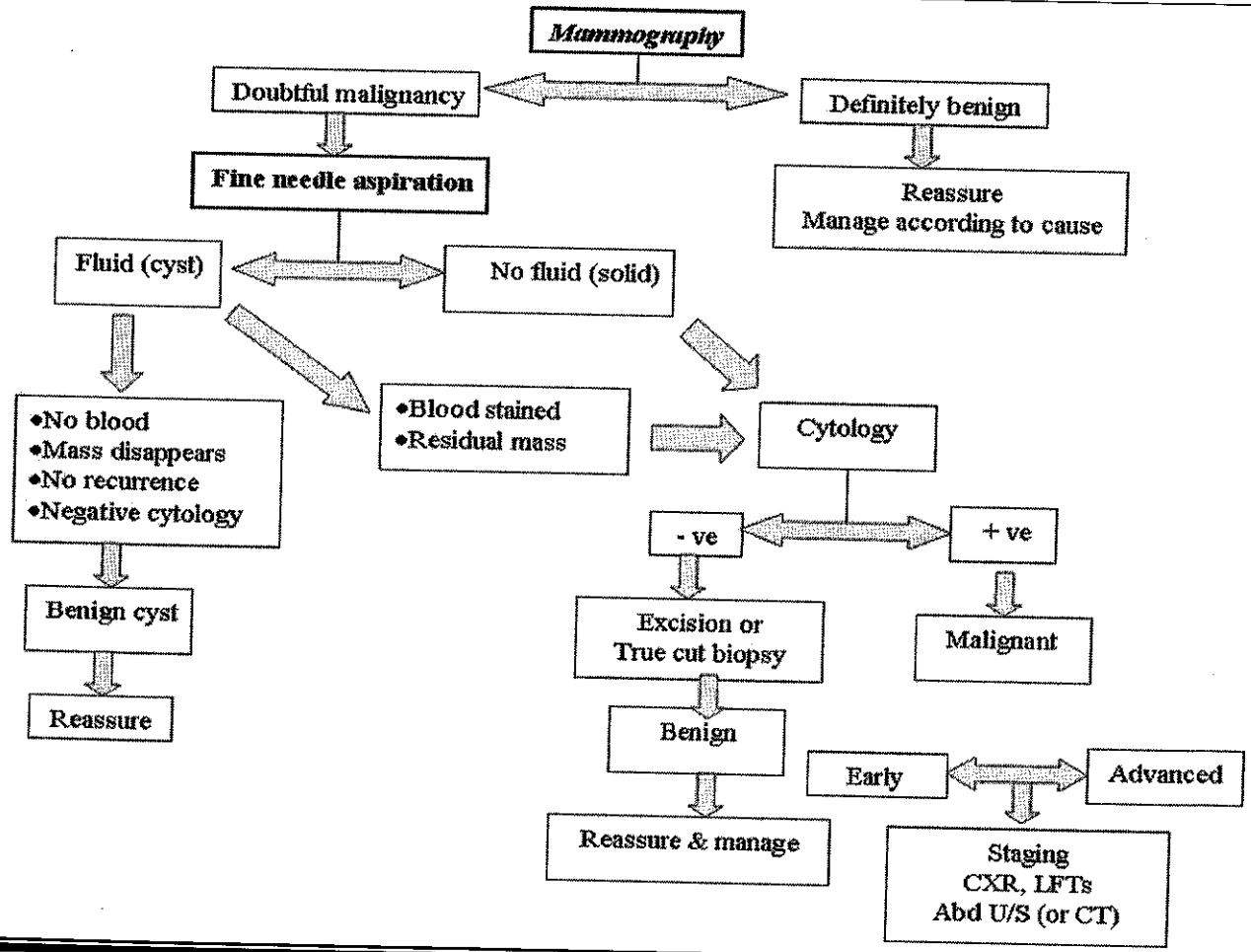
Prognosis:

➧ **Prognosis of patients with breast carcinoma depends on the following factors :**

1. **The type of the tumor:** The best prognosis is provided by the in situ carcinoma and Paget's disease, while the worst is the inflammatory carcinoma.
2. **The T stage of the primary tumor:** The higher the T stage, the worse is the prognosis.
3. **The site of the tumor:** Medial half tumors have a worse prognosis than those of lateral half due to early involvement of the internal mammary lymph nodes.
4. **The involved lymph nodes :** Size, mobility, number and location of the involved lymph nodes.
   Assessment of lymph node involvement depends on the histological examination :
   (a) Large fixed nodes are of bad prognosis.
   (b) The number of involved nodes largely affects the prognosis :
   - Patients with negative axillary nodes have a 10-year survival rate of 65 %.
   - Patients with 1-3 positive axillary nodes have a 10-year survival rate of 38%.
   - Patients with more than 4 positive axillary nodes have a 10-year survival rate of 13%.

   (c) The prognosis worsens the higher the level of the affected nodes in the axilla. High-level nodes carry a bad prognosis.
5. **The presence of distant metastasis**
6. **Hormone receptor status: Receptor positive tumors respond more often to hormonal therapy and have a better prognosis than those that are receptor negative.**

Investigations of clinically doubtful breast mass



Diseases of the male breast

Gynecomastia

Definition : Generalized enlargement of the male breast.

Etiology :

[A] Physiological :

1. **Infantile gynecomastia :** Due to circulating maternal sex hormones.
   - It usually resolves within six months.
2. **Pubertal gynecomastia :**
   - It occurs in up to 70% of normal pubertal boys.
   - It is often asymptomatic.
   - It usually resolves within 2 years.
3. **Senile gynecomastia :** Due to the reduction of testicular function.

[B] Secondary gynecomastia :

1. Reduced production of testosterone e.g. after orchidectomy, testicular atrophy.
2. High levels of estrogen caused by feminizing testicular tumors and suprarenal tumors.
3. Failure to metabolize estrogen in cases of chronic liver disease.
4. Drugs as estrogen, cimetidine, digoxin, spironolactone, phenothiazines. and cannabis.
5. Ectopic hormonal production in bronchogenic carcinoma.

Clinical features :

- **The usual complaint is breast enlargement** which may be unilateral or bilateral, and with or without tenderness.
- **Examination reveals a subareolar mass (a disc of tissue),** which is soft and mobile. Any induration, or fixation should raise the suspicion of malignancy (this is not a possibility in children and adolescents).
- **Examination should include the abdomen and testes.**

Investigations : These are not required for neonatal and adolescent cases.

1. Liver function tests.
2. Biopsy in suspected cases of malignancy.

Treatment :

- **The neonatal and adolescent types :** usually resolve spontaneously.
- **Secondary gynecomastia :** treatment of the underlying condition.
- **Persistent gynecomastia :** can be treated surgically by subcutaneous mastectomy.

Carcinoma of the male breast

- **Incidence :** male : female carcinoma = 1:100.
- **Staging & treatment :** similar to that for carcinoma of the female breast, but castration is the principal mean of hormonal control of advanced cases.
- **Prognosis :** is worse than that of female breast cancer as it rapidly becomes attached to the skin & chest wall.

Differential Diagnosis of Breast Pain

1. Itching pain :	a. Ordinary eczema. b. Scabies.
2. Burning pain :	Cracked nipple.
3. Acute dull pain :	a. Milk engorgement. b. Acute mastitis.
4. Throbbing pain :	Acute abscess (suppuration).
5. Dull ache related to menstruation :	Fibroadenosis.
6. Carcinoma is painless, rarely painful from :	a. Associated abscess. b. Nerve infiltration. c. Mastitis carcinomatosa.

Differential diagnosis of retracted nipple

[A] Congenital :

- Present since childhood. Nipple fails to develop.
- It is due to failure of the nipple to follow the growth of the breast.
- The retracted nipple possess difficulty only when the lady is lactating, and is a predisposing factor for the development of mastitis. The problem may be spontaneously corrected during pregnancy or lactation.

[B] Recent :

- Nipple was normal. Retraction resulted from excess fibrosis in :

1. **Carcinoma :** it is the most important cause.
2. **Chronic inflammation :** Chronic abscess, mammary duet ectasia and tuberculosis.
3. **Fibroadenosis :** rare.

Causes of huge breast

1. Sarcoma.
2. Mastitis carcinomatosa.
3. Encephaloid carcinoma.
4. Colloid carcinoma.
5. Soft fibroadenoma.
6. Diffuse massive hypertrophy.

Face & Mouth

Face & Mouth

Development of the face

The face is formed of 5 processes :

1. Frontonasal process. 2. Two maxillary processes. 3. Two mandibular processes.

Congenital anomalies of the face

[A] Abnormalities due to failure of fusion :

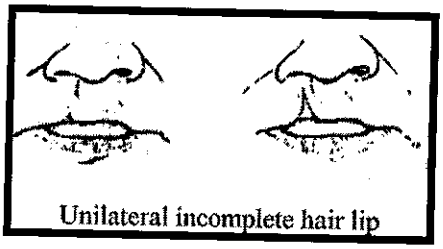
1. Facial clefts :
   (a) Craniofacial clefts. (b) Cleft lip. (c) Cleft palate.
   - The last two defects are the most common congenital anomalies of the head (1:700 live births).
2. Preauricular sinus.
3. Macrostomia.

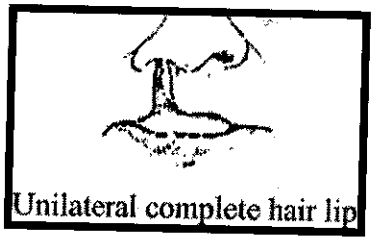
[B] Abnormalities due to excessive fusion :

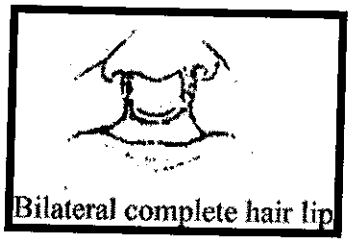
1. Microstomia.
2. Narrow palpebral fissure.

[C] Dermoid cyst.

Cleft Lip







Etiology :

➧ Failure of fusion between the median process of the frontonasal prominence and one (unilateral) or both (bilateral) maxillary processes.

Etiological factors include :

1. Familial disease due to genetic susceptibility.
2. Consanguinity.
3. Prenatal exposure to alcohol, anticonvulsants, X-ray or virus infection.
   - **Cleft lip may be associated with other congenital anomalies in up to 35 % of cases.**

Pathology : (Types)

1. Unilateral 85 % or **bilateral** 15 %. Rarely median and very rarely in the lower lip.
2. Complete (reaching the nostril floor) or **incomplete.**
3. The cleft lip may be simple or complicated (if it is associated with cleft palate or alveolus).

Effects on function :

1. Cleft lip does not interfere with suckling, but may be associated with abnormal teeth growth.
2. Psychological upset of the parents.

Management :

Operation is best performed at the age of 3-6 months **(before teething).** The infant should be at least 10 pounds in weight and the hemoglobin level should be at least 10 gm %.

Principles of the operation :

1. Paring of the edges.
2. The lip is separated from maxilla to make the lip lax.
3. Suturing the 3 layers of the lip (skin, muscle, and mucous membrane).
4. In bilateral : The repair is done either as one stage or as two-stage operation.

(Preliminary orthodontic treatment might be indicated especially in protruded premaxilla).

Cleft Palate

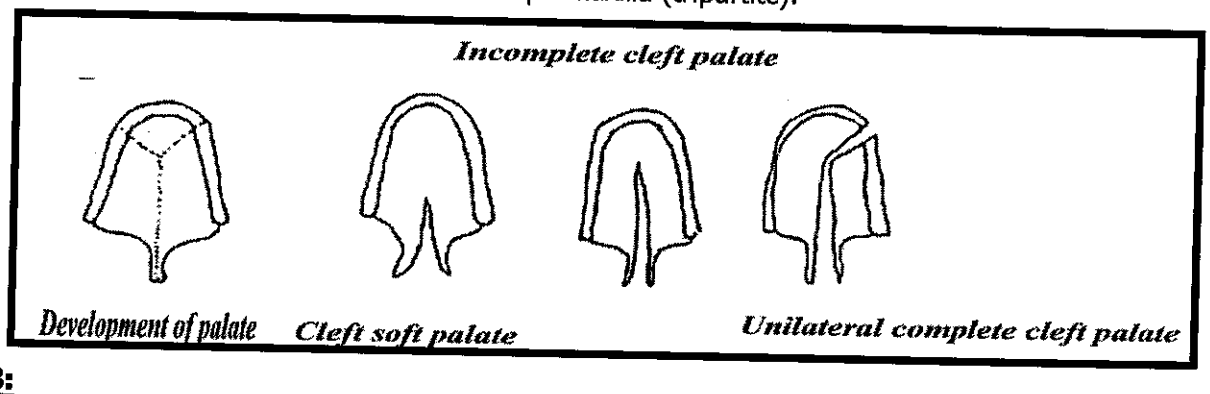
Etiology :

Due to incomplete fusion between the two palatal processes of the maxilla with the nasal septum and possibly the premaxilla (from the frontonasal process).

Predisposing factors : See cleft lip.

Pathology : (Types)

1. Cleft uvula.
2. Cleft soft palate.
3. Cleft soft and hard palate (complete).
4. Complete cleft palate plus one side of premaxilla (bipartite).
5. Complete cleft palate plus the two sides of premaxilla (tripartite).



N.B:

- **The cleft of the primary palate :** A cleft in the structures anterior to the incisive foramen.
- **The cleft of the secondary palate:** A cleft in the structures posterior to the incisive foramen.
- Associated with cleft lip in 50 % of cases.
- May be associated with other congenital anomalies.

Effects on function :

1. Impairment of normal suckling, due to inability to create a negative intraoral pressure.
2. Regurgitation predisposes to aspiration pneumonia.
3. Recurrent otitis media, which may lead to hearing loss.
4. Speech defects secondary to : (a) Nasal tone due to nasal-oral communication (b) Hearing loss.
5. Interference with normal teething.

Management :

Timing of the operation : • At 12-18 months (before phonation).

Preoperative management :

1. Attention to feeding. As there is inefficient breast-feeding, a bottle with a large hole is used or spoon-feeding is used in an upright position.
2. Attention to chest troubles.

Principles of surgery :

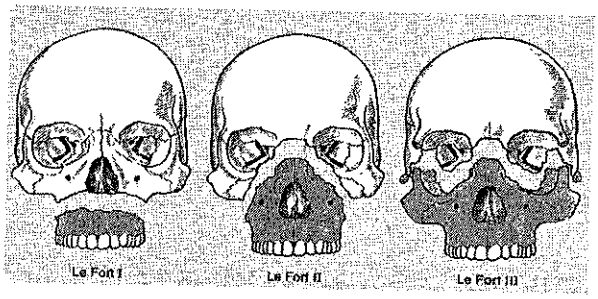
1. Paring of the edges.
2. Lateral relaxation incisions are needed.
3. Suturing is done in 3 layers in the middle line (nasal mucosa, muscle layer then oral mucosa).
4. Fracture of the pterygoid hammulus to relax the tensor palati.
5. Pharyngoplasty may be added to narrow the nasopharyngeal isthmus.

Post-operative follow up :

1. Speech therapy.
2. Tonsillectomy and adenoidectomy are better avoided.
3. Orthodontic treatment.

Maxillofacial injuries

1. **The maxillofacial region can be divided into 3 parts:**
   - The upper face: the frontal bone and frontal sinus.
   - The mid-face : the nasal, ethmoid, zygomatic and maxillary bones.
   - The lower face: the mandible.
2. Maxillofacial trauma is usually caused by: RTA & Falls
3. X-ray and CT scanning provide the mainstay of fracture investigation.
4. **Specific fractures**



1-factures of maxilla

☒ Presented by pain, salivation, mal-occlusion, oedema, epistaxis, diplopia and crepitation.

☒ **Classification:**

Le Fort I 1- A transverse fracture above level of the teeth.
- Treated by inter-maxillary fixation.

Le Fort II 1- A pyramidal-shaped fracture.
2-fracture transverses base of nose through posterior wall of maxillary antrum & across orbit.
- Treated by intermaxillary fixation & fixed by wires to zygomatic process of frontal bone.

Le Fort III 1-There is a craniofacial dysjunction.
- Treated by correction of nasal & zygomatic fractures. Then the same as in Le Fort II.

2-fractures of the mandible

Etiology : traumatic or pathological.

Site : Fractures may occur in body (commonest), symphysis, angle, ramus, coronoid or alveolar process

Clinical picture :
- As general.
- Are usually compound into the mouth because the mucoperiosteum is firmly attached to the bone.
- **Coleman's sign :** swelling, hematoma in the floor of the mouth.
- There is irregularity of the line of the teeth.

Investigations : Plain x-ray is diagnostic.

Treatment :

[A] First-aid treatment : the jaw is supported, analgesics, antibiotics & oral hygiene.

[B] Reduction & fixation :
- If there is displacement, reduction is done under anesthesia.
- Fixation for 3 weeks is done by :

1. Interdental wire.
2. Arch bars.
3. Direct intermaxillary wire.
4. Interosseus wire.
5. In edentulous patients, the mandible is fixed by the use of fixed artificial dentures.

3-Soft tissue injuries:

- facial nerve injures medial to midpupillary line need NO treatment but if lateral requires nerve repair
- Parotid duct injures end-to-end anastomosis over a small silastic catheter.
- Lips injures should be sutured in 3 layers with respect to the anatomical landmarks
- Nasal injuries are sutured in 2 layers. evacuation of septal hematoma to avoid saddle nose deformity
- Intraoral injuries: edges are derided and loosely approximated

Dangerous area of the face

Definition: The area of the face between the lines passing from the outer canthus to the angle of mouth

Importance: Infection in this area is liable to cause cavernous sinus thrombosis.

How does infection reach the cavernous sinus ?

- The angular vein communicates with the ophthalmic veins which drain into the cavernous sinus.
- The anterior facial vein communicates with the cavernous sinus via an emissary vein.

Carcinoma of the face

Lip carcinoma

It is a squamous cell carcinoma of the skin of the lip. It is well differentiated **(Grade I and II according to Broder's classification)** with the formation of cell nests.

Precancerous lesions : (All start by letter S)

1. **Simple tumor :** Squamous cell papilloma.
2. **Sepsis :** In oral cavity as dental ulcer.
3. **Smoking** (especially tobacco pipes).
4. **Syphilis.** **5. Spices.** **6. Sun rays** (especially U.V. rays).

Clinical picture :

- **More in men.** • **Age :** Above 60 years.
- **Site :** The lower lip is affected in 93 %, upper lip 5 % and the angle of the mouth 2 % (the last one is more malignant and infiltrates both lips).
- **Size :** Variable.
- **Shape :** Either malignant ulcer (commonest), malignant fissure, malignant nodule, malignant cauliflower mass or diffuse infiltrating type giving woody lip.
- **Edge :** Of the ulcer everted. • **Margin :** Indurated.
- **Floor :** Malignant tumor tissue. • **Base :** Indurated.
- **Discharge :** Blood.
- **Lymph nodes :** Stony hard and painless, at first mobile but later become fixed.

Complications :

1. Spread :

- Local : involving the whole lip, mandible, maxilla or gum.
- Lymphatic spread :
  (a) Lower lip to the submental and submandibular and upper deep cervical.
  (b) Upper lip to preauricular then deep cervical.
  (c) Angle to both groups of lymph nodes (so the poor prognosis of tumors of the angle).
- Blood stream : Late.

2. Secondary infection. **3. Hemorrhage.**
4. Aspiration pneumonia. **5. Difficulty in eating and in speaking.**

Treatment :

[A] Irradiation : of the primary and block dissection of secondaries.

- **Irradiation either by deep X-ray therapy or radium needles or mould.**

[B] Surgical excision : This is indicated in : **(SBR)**

1. **S**mall suspicious nodule or ulcer. To be sure of its pathology excision biopsy is essential.
2. Cancer on top of **S**yphilis as there is endarteritis decreasing the blood supply to the area. Irradiation effect depends on decreasing blood flow to the tumor, which is already decreased.
3. If the **B**one is infiltrated.
4. In **R**ecurrence after irradiation or **R**adio-resistant.

Surgical management of the primary :

- V-shaped excision of lip is done. If the mandible is infiltrated central hemimandibulectomy is combined with V shaped excision of the lip.

[C] Treatment of lymph nodes : (Only 10 % of patients have lymph nodes when first seen)

1. If there is no lymph node enlargement : Monthly follow up, up to six months.

2. If there is lymph node enlargement : It may be due to secondary infection. Wait for one month after that period if they are due to metastasis, suprahyoid block dissection of lymph nodes is done, in which submental, submandibular, upper deep cervical lymph nodes on both sides are removed as one mass **(en block).**

Cystic swellings of the face

1. External angular dermoid.
2. Meibomian cyst.
3. Mucocele of the lacrimal sac.
4. Meningocele.
5. Sebaceous cyst.

Items	Sebaceous cyst	Dermoid cyst
Cause	Retention	Sequestration, implantation or teratomatous
Site	**Any site especially :** face, scalp and scrotum. **Never in palm & sole.**	**Special site :** line of fusion in face, midline & palm.
Shape	Hemispherical	Complete sphere
Attachment to skin	At one point	Not attached
Age	Adult	Dates since birth or childhood

Cystic swellings of the floor of the mouth

[1] Mucous cysts of minor salivary glands.

[2] Ranula :

- It is a bluish painless swelling on one side of the floor of the mouth in the angle between the tongue and the floor. The swelling is cystic in consistency, translucent in appearance. The duct of the submandibular salivary gland is stretched over its surface. It may have a deeper prolongation in the neck in the submandibular region **(plunging or Thompson's ranula).**

The wall is lined by : columnar epithelium and the contents are clear gelatinous material.

Origin : This is a retention cyst arising from a sublingual salivary gland or an extravasation cyst arising from a damaged sublingual gland.

Treatment : Excision is very difficult (as the wall is thin and adherent). Partial excision of the roof, the edges of which are sutured to the mucosa lining the floor of the mouth **(marsupialization)** is the treatment of choice **(i.e. the floor of the ranula will form part of the floor of the mouth).**

[3] Sublingual dermoid cyst :

- It is a sequestration dermoid cyst occurring in the midline either supramylohyoid appearing in floor of mouth or inframylohyoid appearing in the neck. It is lined by squamous epithelium and contains sebaceous material. The wall is thick. It is not translucent **(D.D. ranula).**

- **Treatment :** Supramylohyoid cysts are excised through the floor of the mouth. Inframylohyoid cysts are excised through a curved submandibular incision.

The Tongue

Tongue injuries

Treatment of tongue injuries :

- Bleeding is arrested by hooking the tongue forward with a finger and compressing it against the mandible. Then the lacerations could be sutured in the operating room.

Glossitis

[1] Acute superficial glossitis :

- Pyogenic infection of the tongue is extremely rare.
- Gross swelling of the tongue due to insect bite or angioneurotic edema may need nasopharyngeal airway insertion or a tracheostomy.
- Minor degrees of glossitis can occur due to scalding fluids, sepsis, diabetes, and alcoholism- This form presents by a painful red glazed tongue.

[2] Chronic superficial glossitis : Due to chronic irritation. It occurs in middle and old ages.

Pathology :

- The disease affects the anterior two thirds of the tongue.
- There is hypertrophied papillae, leukoplakia, red glazed tongue, cracks, fissures and warty projections.
- Chronic superficial glossitis is precancerous.

Treatment : 1. Elimination of the irritating source.
2. Mouth wash. 3. In resistant cases, excision of the lesion.

[3] Tuberculous glossitis :

- Due to autoinoculation from pulmonary tuberculosis, through the sputum. The lesion presents either as a circumscribed interstitial tuberculoma or as shallow oval painful ulcers with overhanging margin.

[4] Syphilitic glossitis : The tongue may be affected in any of the 3 stages of syphilis :

1. Primary stage : extragenital painless chancre at the tip of the tongue with enlargement of the submandibular lymph nodes.

2. Secondary stage : Multiple shallow snail track ulcers appear on the sides and undersurface of the tongue. Mucous patches are present, Hutchinson wart (condyloma) may occur in the midline.

3. Tertiary stage : gumma or chronic superficial glossitis. A gumma presents as slowly growing midline swelling get necrosed and separated as wash leather sloughs leaving a deep crater like-ulcer. A gumma has to be differentiated from carcinoma which usually does not occur in the midline and which produces tethering of the tongue.

Ulcers of the tongue

I- Traumatic :

1. Dental ulcer :

- **Site :** Margin of the tongue.
- **Size :** Usually small.
- **Shape :** Circular.
- **Pain :** Usually present.
- **Lymph nodes :** Chronic inflammatory enlargement.
- **Treatment :** Removal of the septic tooth or denture is followed by healing of the ulcer.

2. Frenular ulcer : (Post-Pertussius ulcer)

- **Secondary to whooping cough** due to severe protrusion of the tongue during cough.
- **Site :** Inferior surface of the tongue at the frenulum.
- **Size :** Small.
- **Surface :** Granulating.
- **Edge :** Sloping.
- **Base :** Not indurated.

II- Inflammatory :

[A] Acute :

1. Dyspeptic ulcer:

- **Etiology :** unknown.
- **Site :** The sides of the tongue and its dorsum and inner surface of lip and cheek.
- **Size :** Small.
- **Surface :** Covered with white scabs.
- **Base :** Not indurated.
- **Edge :** Sloping.
- **Margin :** Hyperemic.
- **Number :** Multiple.
- **Treatment :**

(a) Of the cause. Paint ulcer with gentian violet.
(b) Antiseptic mouth wash, alkaline lotion & anesthetic gel.

2. Herpetic ulcer : Painless, multiple, small ulcers.

3. Lichen planus : This is supposed to be due to an autoimmune mechanism. It affects the skin and oral mucosa and may cause ulcers plus hyperkeratotic lesions.

[B] Chronic :

1. TB ulcer : This type of ulcer is secondary to open pulmonary TB.

- **Site :** Dorsum of the tongue posteriorly and the tip of the tongue anteriorly.
- **Size :** Usually small.
- **Number :** May be multiple.
- **Floor :** Pale granulation tissue with multiple tubercles.
- **Edge :** Undermined.
- **Base :** Not indurated.
- **Pain :** Severe.
- **Lymph nodes :** TB lymphadenitis. Chest X-ray may show pulmonary T.B.
- **Treatment :** Anti-TB treatment, oral hygiene.

2. Syphilitic ulcer : Due to breakdown of a gumma.

- Can occur in the secondary stage as snail track ulcers or in the tertiary stage as a gammatous ulcer.
- **Site :** Dorsum of the tongue in the midline.
- **Size :** Large.
- **Shape :** Rounded or circinate.
- **Edge :** Punched out.
- **Floor :** Wash-leather.
- **Base :** Indurated.
- **Pain :** It is painless.

3. Chronic superficial glossitis :

- There is hyperkeratosis forming a white patch of thickening like smooth white paint **(leukoplakia).**
- The epithelium may be shed out forming **row beef tongue.**
- Later **submucous fibrosis** occurs leading to **fissure formation.**
- Associated **endarteritis obliterans** in the blood vessels deep to submucous membrane.
- **These changes are precancerous.**

Treatment : Care for oral hygiene. If leukoplakia is localized, excision is preferred.

III- Neoplastic ulcers : Squamous cell carcinoma (discuss).

Carcinoma of the tongue

Incidence :

- **Age :** More above age of 60.
- **Sex : male** to female ratio used to be 10:1. Now, there is decrease in this ratio. Reduced male incidence due to improved oral hygiene, efficient treatment of syphilis. Increased female incidence due to increased smoking among females.
- **Geographical :** The disease has a high incidence in India because of increased tobacco chewers & use of spicy foods.

Etiology :

Predisposing factors :

Smoking, sepsis, spices, spirits, sharp teeth, clay pipe and bad oral hygiene.

Precancerous lesions are :

1. Syphilis.
2. Dental ulcers.
3. Chronic superficial glossitis.
4. Papilloma of the tongue.
5. Leukoplakia : It shows hyperkeratosis and possibly dysplasia.
6. Erythroplakia : This is deep reddening with mucosal atrophy.

Pathology :

Squamous cell carcinoma : (contributes in 90 % of cases)

- **Site :** Lateral margin of the anterior two thirds (50 %). Posterior third 20 %.
  Less common sites are the ventral surface and the dorsal surface. The tip is rarely affected.

Naked eye appearance : It is either :

1. Malignant ulcer : See carcinoma of the lip.
2. Raised oval plaque.
3. Hard submucosal nodule.
4. Deep indurated fissure.
5. Diffuse infiltrating tumor (woody tongue) is rare.

- **The surrounding mucous membrane may show leukoplakia.**

Microscopic appearance :

1. Squamous cell carcinoma (less differentiated than that of the lip). Posterior 1/3 carcinoma is less differentiated.
2. Adenocarcinoma from the minor salivary glands is rare.

TNM staging :

T_0	No evidence of tumor.
Tis	Carcinoma in situ.
T_1	< 2 cm.
T_2	2-4 cm.
T_3	> 4 cm.
T_4	Involvement of the base.
N_0	No lymph nodes.
N_1	Ipsilateral single node < 3 cm.
N_{2a}	Ipsilateral single node 3-6 cm.
N_{2b}	Ipsilateral multiple nodes < 6 cm.
N_{2c}	Bilateral or contralateral nodes < 6 cm.
N_3	Lymph nodes > 6 cm.
M_0	No metastasis.
M_1	Distant metastasis.

Spread of the disease :

1. Direct spread : To the floor of the mouth, mandible. Tumors of the posterior third spread to the tonsils, pharyngeal wall and larynx.

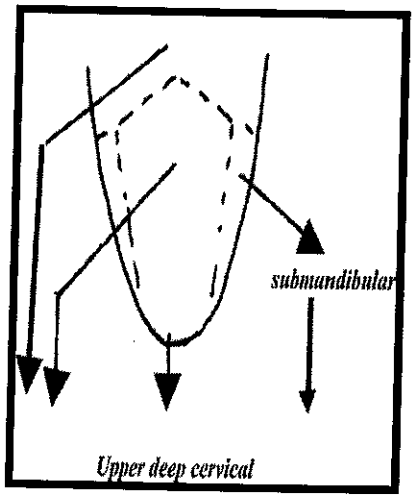
2. Lymphatic spread : Unlike squamous cell carcinoma of the lip, carcinoma of the tongue disseminates early to the lymph nodes of the neck :

(a) Tip : Spread from the tip of the tongue to the submental lymph nodes then to both submandibular and to both upper deep cervical nodes on both sides.

(b) Anterior 2/3 : Tumors of the lateral third disseminates to the ipsilateral submandibular and then to the upper deep cervical lymph nodes. Those near the midline disseminate bilaterally.

(c) Posterior 1/3 : Spread occurs directly to the upper deep cervical lymph nodes.

3. Blood spread : This is more for tumors of the posterior third.



Clinical picture :

- **Age :** > 60 years.
- **Sex :** more in males.

- **In early cases The patient may complain of :**

1. **Ulcer with indurated base and everted edges.**
2. **Deep indurated fissure.**
3. **Indurated mass.**

- **Late presentation :**

1. Pain in tongue : The pain is referred to the ear via the auriculotemporal nerve.
- ✓ Pain, early due to infection, later due to lingual nerve infiltration.

2. Excessive salivation : which may be blood stained.
➡ The classic picture of tongue cancer is that of an old man sitting in the outpatient clinic with cotton wool in his ear, and blood stained saliva drippling from the mouth.

3. Dysphagia : especially in posterior one third.
4. Fetor : Due to necrosis and infection.
5. Inability to speak (dysarthria).
6. Secondaries in the neck.

Complications :

1. **Inhalation of necrotic tissue** leading to bronchopneumonia.
2. **Combined cancer cachexia and starvation** due to pain and dysphagia.
3. **Hemorrhage.**
4. **Spread.**
5. **Asphyxia** may occur.

Investigations :

1. **Biopsy :** from the edge of the ulcer.
2. **Fine needle aspiration cytology :** of suspected cervical lymph nodes.
3. **CT of neck and mandible.**

Treatment :

[A] Radiotherapy : is indicated in :

1. Early cases.
2. In tumors of posterior 1/3.
3. In inoperable cases.

- **Irradiation can be performed by iridium needles or by external beam radiotherapy.**
  - ✓ Radiotherapy is avoiding the disfiguring side effects of surgery.
  - ✓ Mucositis, dysphagia & osteo-radionecrosis are disadvantages of Radiotherapy.

[B] Surgical treatment : For operable cases it is indicated in :

1. **S**mall **S**uspicious nodule.
2. Cancer on top of leukoplakia or **S**yphilis.
3. Tumor infiltrating the mandible (**B**one).
4. **R**ecurrence after irradiation.

- **Surgical removal is as follows :**

1. **Carcinoma in situ:** excision with 1 cm safety margin on the sides and 1/2 cm safety margin in depth.
2. **Carcinoma of the anterior 2/3 of the tongue:** excision with 1 1/2 cm safety margin.
   - a) **For the tip of tongue :** V-shaped partial glossectomy is done.
   - b) **For margin of the tongue :** hemiglossectomy removing one half of the tongue is done.
   - c) **Midline tumors:** total glossectomy is done removing the whole tongue.
   - d) **Tumors of posterior 1/3:** either total glossectomy(difficult)or external irradiation.
   - e) **If the tumor infiltrates the mandible : Commando operation** is done **(combined mandibulectomy and neck dissection operation).** Aiming at removing the lesion in continuity with lymphatic drainage. **Preliminary tracheostomy is needed.**

- ✓ Preoperative irradiation by 4000 rad may be advised.
- ✓ T1&T2 (Less than 4 cm) may equally benefit from surgery or radiotherapy.

[C] Treatment of lymph node metastasis :

1. If lymph nodes are not enlarged or are enlarged and not fixed : Prophylactic or curative block dissection on one or both sides is done.
2. If lymph nodes are enlarged and fixed : Palliative deep X-ray therapy is done.

[D] Palliation is indicated for :

1. Unresectable primary growth.
2. Fixed lymph nodes in the neck.
3. Distant metastasis.

- **Methods of palliation are :**

1. Full dose of therapeutic irradiation is recommended.
2. Palliative resection of the primary if possible may comfort the patient.
3. Analgesics, nasogastric feeding or tracheostomy may be required.
4. Chemotherapy.

Prognosis : (Prognostic Factors)

1. Lymph node involvement is the most important prognostic index.
2. TNM staging.
3. The degree of tumor differentiation.
4. Combined surgery and radiotherapy improve prognosis.
5. Prognosis is better in females.

Swellings related to the jaw

1. Epulis (arising from the gum) as : Fibrous, myeloid, granulomatous, hemangiomatous and malignant epulis.

2. Odontomes (dental origin) as : Dental cyst, dentigerous cyst, and adamantinoma.

3. Bone tumors : as ivory osteoma, osteoclastoma, maxillary antrum carcinoma, fibrosarcoma and osetosarcoma of the mandible.

Epulides

- **Any swelling situated on the gum, e.g.:**

1. Fibrous epulis :

- It is a soft fibroma arising from mucoperiosteum. It is formed of fibrous tissue with spindle cells and plasma cells.
- The swelling is well defined, pedunculated and covered with intact mucous membrane.
- The swelling appears between 2 teeth.
- **Treatment :** Extract a tooth on either side of the tumor and remove wedge of bone with its mucoperiosteum.

2. Granulomatous epulis :

- It consists of excessive granulation tissue in response to chronic irritation either by tooth or irregular denture. The swelling is soft in consistency, bleeds easily on touch, red in color and is not covered by epithelium.
- **Treatment :** Removal of the cause. Cauterization either by diathermy or silver nitrate of this granulation tissue.

3. Hemangiomatous epulis :

- Submucous hemangioma of the gum. Blue in color, compressible, sessile, covered with intact mucous membrane. It is liable for severe bleeding.
- **Treatment :** Injection of sclerosing material or surgical excision.

4. Myeloid epulis :

- It is an osteoclastoma arising in the alveolar margin and erupting through a tooth socket.
- It consists of giant cells and spindle cells together with fibroblasts and fibrous tissue.
- Clinically it forms a sessile smooth swelling covered with intact mucous membrane and fixed to underlying bone.
- Adjacent teeth are loosened.
- **Treatment :** Wide excision with the part of the bone carrying the epulis.

5. Carcinomatous epulis.

6. Sarcomatous epulis: it may occur on top of a fibrous epulis. It is treated by wide excision with the underlying bone.Radiotherapy has no role in treatment of carcinomatous epulis.

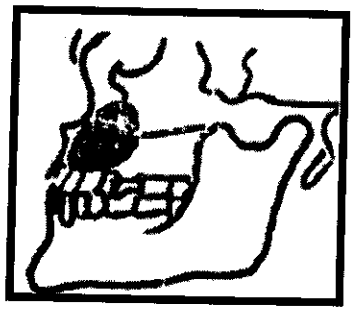
Odontomes

Definition :

Tumors of the jaw arising from tissues involved in the development of teeth.

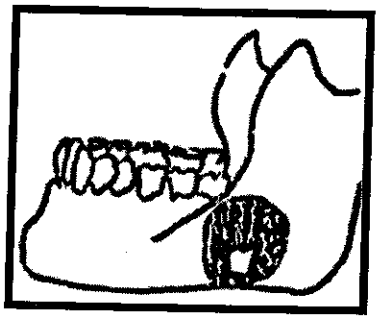
- The tooth has two different origins; first ectodermal from the dental plate and secondary from mesodermal origin **(mesodermal papilla).** Between the developing teeth fragments of ectoderm may persist as the **paradental debris of Malassez** (this may be responsible for tumor formation later on).
- Odontomes may arise from ectodermal or mesodermal origin of a tooth or from both. Only those of epithelial origin are common (dental, dentigerous cysts and adamantinoma).

Dental cyst



- Cystic swelling related to the root of a normally erupted tooth **(usually incisor tooth of the upper jaw).**
- It is due to proliferation of paradental epithelial debris due to chronic irritation by an infected tooth.
- It is unilocular, slowly growing, lined with epithelium and filled with brownish fluid.
- **Clinically :** It occurs in adult life. The swelling is at first stony hard but later the bone may be thinned out so that **"egg-shell crackling"** can be felt. The tooth related to the swelling is grayish blue in color.
- **Treatment :** Extraction of the affected tooth and excision of the wall of the cyst.

Dentigerous cyst



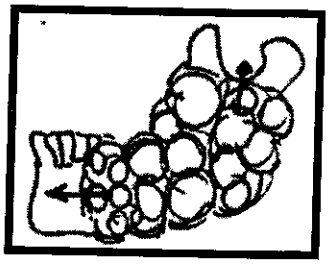
- A cystic swelling related to the root of non-erupted tooth, which is contained into the cyst. It usually affects the third molar tooth.
- It occurs in young age. It is commoner in the mandible than upper jaw.
- The mandible is expanded uniformly and sometimes produces eggshell crackling. The cyst grows slowly. It is lined with epithelium and filled with glory fluid around a non-erupted tooth.
- **Treatment :** Remove the roof of the cyst and the lining membrane. If the tooth is normally directed and expected to grow upwards normally, it is not removed. If the tooth is maldirected, this tooth is removed.

Adamantinoma (Eve's tumor)

- It is a basal cell carcinoma arising from the epithelial debris of Malassez. The tumor arises at the angle of the mandible. It grows slowly forwards into the body of the mandible and upwards into the ascending ramus. It consists of cystic and solid areas separated by fibrous and bony septa. The cystic areas are lined by squamous and basal cells and contain brownish mucoid fluid in the center. The solid areas are composed of fibrous tissue.

- **Clinically :** It is more in females. Age between 25-45 years. Patient presents with a painless lobulated swelling expanding the mandible on the outer side more than the inner. The consistency is bony hard, but eggshell crackling may be elicited.

- **Investigations :**



1. Plain X-ray shows expanding translucent shadow divided by numerous septa into equal lobules **(honey-comb appearance).**
2. CT scan & MRI.
3. Biopsy.

- **Complications :**

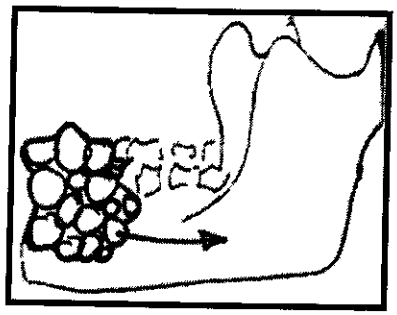
1. Ulceration, infection, bleeding & falling of teeth.
2. Pathological fracture (rare).
3. It is a locally malignant lesion, so it is liable to recurrence after inappropriate surgery. Sometimes it turns malignant.

- **Treatment :** Resection of tumor bearing segment of the jaw with a healthy margin on either sides. The defect is repaired by bone graft.

Bone tumors

As general

Giant cell lesions of the jaw



1. Giant cell granuloma :

- Affects the lower jaw more commonly.
- It arises above the symphysis menti as a soft reddish mass and grows backwards in the body of the mandible, but not upwards in the ascending ramus.
- **Cut section :** Lobulated tumor.
- **Histologically :** there are giant cells, spindle cells and numerous blood vessels.

Clinical picture :

It is a **painless, slowly growing** tumor, **expanding the jaw equally** on both sides (due to presence of osteoclasts). It may show **eggshell crackling.**

Investigations :

Plain X-ray shows expanding translucent shadow with unequal trabeculae **(soap bubble appearance).**

Treatment :

Careful enucleation and gentle curettage. In large tumors, resection of the mandible is done.

2. Osteoclastoma :

It is rare and similar to the previous mentioned lesion, but it is malignant on histological examination.

3. Aneurysmal bone cyst : It forms a soft, sponge like tumor.

Carcinoma of the maxillary antrum

- It is commoner in **males.**
- **Age :** 40 years.
- The tumor is commonly squamous cell carcinoma or columnar cell carcinoma.

- The effect and clinical presentation depends on which wall of the maxillary antrum is involved :

1. Medial wall causes unilateral nasal obstruction, bleeding from nose and epiphora.
2. Roof of the antrum leads to unilateral proptosis and diplopia.
3. Anterior and lateral walls leads to a swelling in face.
4. Floor, bulges in the roof of the oral cavity and may cause pain referred to the teeth.

- **Treatment :** Either radical surgical excision of the upper jaw or irradiation.

Salivary Disorders

Salivary glands

Non-Neoplastic Salivary Gland Diseases

This term includes different disease groups, which are :

1. Congenital diseases : as ectopic parotid tissue, cystic hygroma & sialectasis.

2. Infections:

(a) Acute : viral, bacterial.

(b) Recurrent subacute or chronic.

(c) Rare : T.B.

3. Salivary stones.

4. Salivary fistula.

5. Degenerative diseases (sialectasis).

6. Autoimmune salivary diseases.

7. Drug-induced enlargement of the salivary glands has been reported e.g. iodide containing compounds, Thiouracil, hypotensive drugs & contraceptive pills.

8. Metabolic & endocrinal causes : include liver cirrhosis, diabetes mellitus, alcoholism &malnutrition.

Sialectasis

(Degenerative disease)

Definition : Sialectasis means abnormal dilatation of the small branches of the salivary ducts.

Etiology : Not known, but the childhood type is known to be a familial disease.

Clinical picture : The usual presentation is unilateral recurrent attacks of sialadenitis.

Investigations : Sialography "snow storm appearance".

Treatment :

1. Conservative: Stimulate salivary flow and massage the affected gland with antibiotics during attacks of inflammation.

2. Surgical excision : of the gland.

Autoimmune salivary diseases

1- Mikulicz's disease: Consists of enlargement of the salivary and the lacrimal glands and dryness of the mouth.

2- Sjogren's disease

(a) Primary Sjogren's disease : consists of dryness of the mouth (xerostomia) and dryness of the eye (keratoconjunctivitis sicca).

(b) Secondary Sjogren's disease : as well as rheumatoid arthritis, salivary gland discomfort, but only 30 % have sialomegaly.

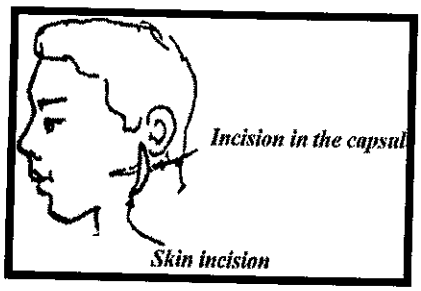
- **Complications :**

Patients with Sjogren's disease are 44 times prone to the development of lymphoma than the general population.

Acute parotitis

Definition :

Acute inflammation of the parotid gland.



Etiology :

Causative agent : pyogenic organisms usually staph. Aureus.

Route of infection :

1. Retrograde infection from the mouth through the duct.
2. Blood born infection.

Predisposing factors :

1. Dehydration.
2. Debilitating medical diseases as typhoid and cholera (dry infected mouth).
3. Obstruction of the parotid duct.
4. As a complication of septicemia.

Pathology :

- As the inflammatory process proceeds, the resultant edema and exudate will increase the tension inside the closed space formed by the parotid capsule and fascia.

Clinical picture :

1. **Severe toxemia.**
2. **A painful swelling** on the side of the face. **Edema** appears early and **fluctuation** appears later.
3. **Pus** can be expressed from the duct of the gland in the oral cavity.

Treatment :

1. Prophylactic : correct dehydration, and care for oral hygiene.

2. Early cases : respond to antibiotic therapy.

3. In fulminating cases : Early decompression of the gland is needed **(even before appearance of fluctuation)**. A curved skin incision is done following the angle of the mandible. The skin flap is reflected. The capsule of the parotid is opened transversely by **Hilton's method** (this avoids injury of facial nerve).

Salivary calculi

Incidence : Stone may occur in the parotid gland, which is very rare, but commonly occurs in the submandibular gland **because:**

1. The parotid secretion is watery while the submandibular secretion is viscid.
2. Drainage of the parotid gland is free, while the submandibular duct ascends upwards towards the floor of the mouth making drainage inadequate.
3. Foreign particles are liable to lodge in the submandibular duct.

Pathology :

- The stone may be single or multiple occurring in the duct or in substance of the gland.
- The stone is formed of calcium and magnesium phosphate and carbonate (radio-opaque).

Clinical picture :

1. Attacks of pain and swelling : in the region of the submandibular gland during meals. Pain is colicky in nature and may be referred to the ear.

2. On examination : The gland is enlarged and tender. Stone in the duct can be felt or even seen in the floor of the mouth.

3. The duct opening : Is congested and pus may be expressed from it.

Investigations: X-ray : a stone in the submandibular gland is radio-opaque in 80% of cases and best seen on a plain occlusal view. More parotid stones are radio-lucent.

Treatment :

1. Stone at orifice is removed through meatotomy.
2. Stone in the duct is removed by cutting directly over it.
3. Stones in the gland are treated by total excision of the gland (sialadenectomy).

N.B:*parotidectomy for calcular disease is difficult as recurrent inflammation makes identification of facial nerve difficult.*

Salivary Neoplasms

Incidence :

Salivary neoplasms constitute 5 % of head and neck tumors. The majority of these neoplasms are benign and most commonly arises in the parotid gland.

Classification :

Salivary Neoplasms

Benign	Malignant
1. Pleomorphic adenoma (mixed salivary tumor)	1. Mucoepidermoid carcinoma
2. Warthin's tumor (adenolymphoma)	2. Adenoid cystic carcinoma (cylindroma)
3. Oncocytoma (oxyphil adenoma)	3. Acinic cell carcinoma
4. Monomorphic adenoma	4. Adenocarcinoma
	5. Carcinoma ex pleomorphic adenoma.
	6. Lymphoma

- **Hemangiomas & lymphangiomas may affect the parotid, but these are pathologically hamaratomas rather than true neoplasms.**

Pathology of benign salivery neoplasm :

1. Pleomorphic adenoma (Mixed parotid tumor) :

- The commonest tumor of salivary glands.
- It occurs in the fourth decade of life, but any age and any sex may be affected.

Pathology :

- It is a pleomorphic adenoma; epithelial cells arranged in sheets, duct like structures, mucoid material (thought to be cartilaginous).
- The tumor is apparently well encapsulated, but the capsule is incomplete (this explain the frequency of recurrence after enucleation).

2. Adenolymphoma = Warthin's tumor :

- **It is an epithelial tumor arising within a parotid lymph node.**
- **Macroscopically :** Multiple cysts of variable sizes are seen.
- **Microscopically :** Epithelial lined spaces in lymphoid stroma.

Pathology of carcinoma of salivary glands :

- It occurs in elderly age, commoner in females.
- It may start de-novo or on top of a mixed salivary tumor.

1. Mucoepidermoid carcinoma :

- Arises from the duct epithelium and is the commonest member of the group.
- It usually affects the parotid.
- Three grades are described; low grade, intermediate grade, or high-grade tumors. The low-grade type is the most frequent and is known to affect children.

2. Adenoid cystic carcinoma (cylindroma) :

- **Histologically** alternating layers of cylinders of cells and hyaline material are arranged around a central cavity forming cylindroma.
- It is the commonest tumor of minor salivary glands.

3. Acinic cell carcinoma :

- The cells of this uncommon tumor resemble the acinic cells of the parotid gland.
- They may metastasize to local lymph nodes or distant sites.

4. Miscellaneous adenocarcinomas :

- They vary according to their histologic pattern.
- They may be trabecular, anaplastic, mucous carcinomas. They are highly malignant.

Clinical picture of benign salivary neoplasm :

Symptoms :

- **Painless, slowly growing swelling in the parotid region.**

Signs :

[A] Inspection shows :

1. Swelling over the angle of the mandible.
2. Elevate the lobule of the ear.
3. The swelling is superficial to masseter muscle.
4. Facial nerve is NOT affected by the tumor.

[B] On palpation :

1. The swelling is not hot, not tender, not attached to the skin, masseter or bone.
2. The swelling is well-defined.
3. Consistency : varies from firm to cystic (but never hard).
4. Pulsation of the superficial temporal artery : is felt normal.
5. Tumors arising in the deeper part of the parotid may bulge in the oropharynx behind the tonsil, hence the importance of examining the mouth cavity.

N.B: ***take care that swelling in the oropharynx may be a carotid body tumor(highly vascular tumor).***

Clinical picture of carcinoma of the parotid :

1. **The tumor forms a rapidly growing swelling, hard in consistency, ill defined and nodular.**
2. **It becomes fixed to skin and deeper structures.**
3. **Local pain, which may be referred to the ear.**
4. **Facial palsy and lymph node enlargement.**
5. **Loss of superficial temporal pulse.**

Differential diagnosis :

1. Extra parotid swellings :

- Lymph nodes (parotid or upper deep cervical), sebaceous cyst & lipomas may resemble pleomorphic adenomas.
- Mandibular, maxillary & infratemporal fossa tumors may produce the false appearance of a parotid enlargement.
- Hypertrophy of the masseter : The condition is sometimes so difficult to differentiate from a true parotid enlargement.

2. True parotid enlargement : that is caused by non-neoplastic salivary gland disease. The gland is diffusely enlarged with no definite lump

Investigations :

Not routine as in most cases clinical diagnosis is reliable enough to proceed to treatment and the pathological diagnosis is not necessary for tumors that behave in a benign way.

1. Biopsy : FNAC is allowed (Reliable investigation which needs expert cytologist).

N.B: ***open surgical biopsy of the major salivary glands is contraindicated.***

2. CT-scan : To show extent of the tumor.

3. Isotope scanning with technetium : Salivary neoplasm shows as a cold spot. Adenolymphoma and oncocytoma show as hot spots.

Treatment :

[1] Tumors that are clinically benign :

- **Enucleation** of a pleomorphic adenoma is easy, but is followed by recurrence that is caused by the left tumor extensions through the defective capsule. The tumor should therefore, be excised with a safety margin.
- The standard operation is **"superficial parotidectomy".** Early in the operation the facial nerve trunk is exposed. All the parotid tissue that is superficial to the nerve and its branches is excised, taking great care not to injure them.
- Tumors arising in the deep part of the gland are treated by **"total conservative parotidectomy"** i.e. excising the whole gland while preserving the facial nerve.
- Benign tumors of the submandibular gland are treated by **"submandibular sialoadenectomy".**

[2] Tumors that are clinically malignant :

- If a pathological diagnosis has not been obtained prior to the operation, **frozen section examination** during surgery is helpful.
- **The standard treatment is radical excision,** which includes wide surgical clearance with cervical lymph node dissection if they were enlarged. For the parotid, this usually necessitates excision of the facial nerve, and probably part of the masseter or the mandible.
- **Radiotherapy** is of limited value, but is administered as a postoperative adjuvant therapy for tumors of high-grade malignancy.

N.B: Salivary neoplasms

1. Carcinoma ex pleomorphic adenoma presents as a painless swelling that has been stationary for years and is now getting bigger.
2. Pleomorphic adenoma of parotid raises the lobule of ear and does not affect facial nerve.
3. Open surgical biopsy of the major salivary glands is contraindicated. FNAC is safer.
4. For tumors of the minor salivary glands of the mouth cavity, excision biopsy is feasible.
5. CT and MRI are the most useful method for assessment.
6. Unlike other salivary neoplasms, adenolymphoma & oncocytoma show a hot spot on isotopic scanning.
7. Even if the facial nerve is preserved in parotid surgery, nerve weakness commonly develops because of neurapraxia that recovers spontaneously few months after the operation.
8. The majority of the tumors arise in the parotid gland are superficial to facial nerve.
9. During submandibular sialadenectomy, 3 nerves can be injured (mandibular division of facial N, inguinal & hypoglossal nerves).
10. The commonest cause of submandibular sialadenectomy is stone in it.
11. For low grade mucoepidermoid carcinoma an attempt to preserve the facial nerve is warrented.
12. For tumors that are clinically malignant, treatment is modified according to the aggressiveness of it.

Swellings in the parotid region

[A] Skin & S.C :

1. Hemangioma. 2. Sebaceous cyst. 3. Lymphangioma.
4. Preauricular dermoid cyst. 5. Melanoma 6. Warts, Boils, Keloids.
7. Lipoma. 8. Hematoma, abscess. 9. Papilloma.

[B] Preauricular L.N.s.

[C] Parotid salivary gland :

1. Inflammation : Mumps, suppurative parotitis, endemic parotitis.
2. Sialectasis. 3. Stones. 4. Tumors.

[D] Masseter : (become fixed on muscle contraction) e.g. Fibrosarcoma or hypertrophy.

[E] Mandible.

[F] Artery : Aneurysm of superficial temporal artery.

[G] Nerve : Neurofibroma.

Swellings in the submandibular region

[A] Skin & S.C. : As before.
[B] Submandibular L.N.s. : Multiple, can be rolled over the lower border of mandible & only felt toward the skin.
[C] Submandibular salivary gland : Single, cannot be rolled over lower border of mandible & felt in the floor of mouth.
[D] Mandible.
[E] Muscle.
[F] Artery : Aneurysm of facial artery.
[G] Nerve : Neurofibroma.

IMPORTANT NOTES:

1-operative treatment of facial nerve injuries:

a) Nerve suture: if nerve graft is needed, the easiest source is the great auricular nerve.
b) If repair is impossible, hypoglossal anastmosis may be done.
c) In hopeless cases, a plastic operation may be done.

2-frey's syndrome:

- It may follow surgery in the region of the parotid gland.
- After injury of auriculo-temporal nerve, parasympathetic fibres from otic ganglion become united to the sympathetic nerves from superior cervical ganglion (cross regeneration).
- Flushing and sweating of the skin innervated by the auriculo-temporal nerve when salivation is stimulated.

Salivary fistula

- Def: fistula that discharges saliva to the skin of the cheek.
- Etiology: injury of the parotid gland or duct resulting from trauma or after surgery.
- Fistula in the gland heals spontaneously.
- Fistula in the duct
  - unlikely to heal on its own (high rate of salivary flow)
  - Sialogram is indicated to role-out distal obstruction.
- Treatment: Surgical treatment is usually necessary.(repair of the fistula or excision of the gland)

Benign lymphoepithelial lesions

- It is uncommon autoimmune disease
- characterized by lymphocytic infiltration & diffuse enlargement of salivary glands particularly parotid & submandibular gland.
- The term "benign" is misleading as 20% of cases develop lymphoma (pre-lymphomatous potential)
- Investigated by Lip biopsy & parotid sialogarm.

Skin & Subcutaneous tissues

Skin & subcutaneous tissues

Sebaceous (epidermoid) cyst

Definition :
A sebaceous cyst is a retention cyst due to blockage of the duct of a sebaceous gland.

Structure :
It is lined by stratified squamous epithelium and contains a foul smelling, white material composed of keratin, epithelial cells and granular debris.

Clinical features :
1. Age : Slowly growing and are, therefore, rarely seen before adolescence.

2. Site : Most commonly in the scalp, face, neck or scrotum but they can occur anywhere except the palm of the sole of the foot, which are devoid of sebaceous glands.

3. On examination:
Small (less commonly large), well defined, cystic swelling, which is usually attached to the skin at one point, which is the site of the duct.
A **punctum** may be seen.

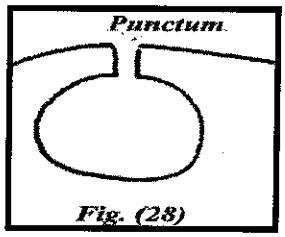


Fig. (28)

Complications :
1. Infection : It may subside by antibiotics, otherwise if an abscess forms, it should be drained and the wall of the cyst curetted. Infection makes the cyst more difficult to excise

2. Sebaceous horn : The contents of the cyst may come out slowly and become inspissated in successive layers over the base.

3. Ulceration : An infected cyst may undergo ulceration leading to the appearance of an ulcer with raised edges. This ulcer is called **(Cock's peculiar tumor)** and it may be mistaken for a carcinoma, but it lacks the induration of malignant lesions. If in doubt a biopsy should be taken.

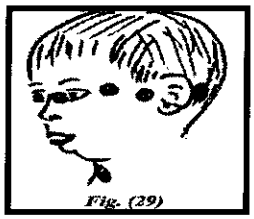
4. Localized alopecia.

Treatment :
Complete excision of the cyst with an ellipse of overlying skin containing the punctum.
The operation can be done under local anesthesia if the cyst is small.

Dermoid cyst

This is a cyst lined by stratified squamous epithelium and contains sebaceous material. Sometimes hairs or sebaceous glands may grow from the wall of the cyst.

Types of Dermoid cyst :

Types	Due to	Sites	Structure
1. Sequestration dermoid  Fig. (29)	Subcutaneous inclusion of portions of the surface epithelium along the lines of fusion of cutaneous segments during fetal life. They may not appear clinically except after few years when the cyst begins to distend.	e.g. external angular dermoids at the outer angle of the eye, at the root of the nose, around the ear or along the midline of the body.	A well-defined, globular, cystic swelling, which is not attached to the skin. The underlying bone may be hollowed out and there may be a pedicle connecting the deep aspect of the cyst to the dura matter.
2. Tubulodermoids	Distension of remnants of embryonic ducts such as branchial cyst.		
3. Inclusion dermoids	Due to inclusion of the epidermis during closure of a cavity.	Sublingual.	
4. Teratomatous dermoid	These are benign forms of teratomas.	Mostly in the ovary but are occasionally found in the testis or posterior mediastinum.	The cyst is lined by squamous epithelium but contains teeth, hairs, bone, cartilage or glands.
5. Implantation dermoids	Puncture wounds which displace some epithelial cells into the subcutaneous tissues. The displaced cells retain their viability and form a dermoid cyst.	Mainly in the fingers, palm or sole.	

Treatment :

The only way of treatment is by **surgical excision.**

NB :

1. In children with a dermoid cyst in the scalp it is better to wait until closure of the skull sutures because some cysts may communicate with the dura.

2. Surgery for a dermoid cyst is more difficult than that for a sebaceous cyst, because a dermoid cyst is deeper.

Vascular anomalies

Classifications:

1-hemangioma
2-vascular malformations
- ✓ Low flow lesions
  - Capillary malformation
  - Venous malformation
- ✓ High flow lesions
  - Arterial
  - Arteriovenous.
  - lymphatic malformations

N.B: differentiation between high and low flow lesions is done by duplex and MRA.

[1] Hemangioma : (previously known as strawberry hemangioma)

1. It affects 10% of white infants and it is three times more common in females.
2. It usually presents by three stages (proliferation, involution & involuted phase).

[2] capillary malformation(port-wine stain)

Items	1. Hemangioma Strawberry angioma	2. capillary malformation Port wine stain (nevus flammeus)
On examination	The lesion is **red** in color and slightly **raised** above the surface.	The lesion is dark purple in color and is **not** raised above the surface. Pressure causes blanching but the color returns immediately after release of pressure.
Site	The commonest site is the face and the head.	May take the distribution of one of the branches of the trigeminal nerve, but the lesion does **not** cross the midline. Sometimes, a port wine stain of the face is associated with similar lesions in the meninges **(Sturge-Weber syndrome).**
Natural history	After the age of one year the lesion starts to undergo involution. Eventually the color fades and flattening occurs so that there may be complete involution at the age of 7 to 8 years. This course is not predictable and sometimes, the lesion continues to grow.	This lesion is present since birth and it does **not** undergo involution.
Complications	The only danger of this lesion is the liability to bleeding if the angioma is in an area liable to friction and ulceration	
Treatment	**(a) Better left for spontaneous involution.** **(b) If problems occur :** Surgery or laser therapies are performed. A course of corticosteroid therapy may help the involution of the lesion.	**(a) Laser ablation.** **(b) Surgery and grafting** may be difficult as the lesion may involve a large area of the face.

[3] venous malformations : previously known as cavernous hemangioma

On examination	Presents as a compressible swelling with some discoloration of the overlying skin.
Site	Occur in the **s**ubcutaneous tissue or the submucous lining of the cheek, lips or tongue. It may involve the internal organs as the liver.
Natural history	The lesion is present **s**ince birth and it has no tendency to involution.
Complications	(a) It may lead to thrombocytopenic purpura due to **s**equestration of a large number of platelets. (b) Atrophy of the overlying skin may lead to **s**evere hemorrhage. (c) **S**epticemia from invasion by microorganisms.
Treatment	(a) Laser therapy. (b) Injection of **s**clerosing solutions as 20 % sodium chloride. (c) **S**urgical excision. *N.B: Compression therapy for venous malformations may relieve pain & edema.*

[4] Arterial malformations (cirsoid aneurysm) :

On examination	The lesion appears as soft, compressible and pulsating swelling with a marked bruit.
Site	It occurs most commonly in the scalp especially in the temporal or occipital regions.
Nature	This lesion is a sort of venous plexus with multiple feeding arteries and so; it is a sort of an arterio-venous fistula.
Complications	Ulceration of the overlying skin may lead to serious hemorrhage, as the bleeding is arterial.
Treatment	Difficult as the swelling is supplied by multiple feeding arteries which have to be ligated. Preliminary embolization of the feeding vessels may be tried.

[5] Arterio-venous malformations:

- It is high flow lesions with an abnormal connections between arteries & veins without intervening capillary bed.
- It may present at birth but may not be evident until late childhood.
- It may be localized or diffuse.
- It may results in growth disturbance or skeletal distortion. There may be local gigantism.

[6] Lymphatic malformations: (see cystic hygroma)

- The commonest cause of congenital macroglossia, macrocheilia & macrotia.

N.B1: Glomus tumor (angioneuromyoma) :

True tumor arising from A-V shunts (glomera) in the distal extremities. It is composed of vascular, neural and smooth muscle fibers. It causes severe paroxysmal pain induced by pressure or temperature changes. It appears as tender purple spot under the nail. Treatment is by excision.

N.B2: Spider nevi and **hereditary hemorrhagic telangiectasia** are considered forms of capillary malformations.

Hereditary hemorrhagic telangiectasia: These are multiple small angiomas in the skin and mucosa with a strong tendency to hemorrhage.

Lipoma

Definition : Benign tumor of the fatty tissue.

Incidence : Common in adult, single or multiple.

Classification :

[A] According to composition :

1. Ordinary lipoma : Mainly fat with little fibrous tissue. Soft.
2. Fibrolipoma : Contains excess fibrous tissue rendering it firm.
3. Angiolipoma : Contains angiomatous tissue rendering it bluish.
4. Myxolipoma : Contains myxomatous tissue.

[B] According to presentation : Lipomas may present as :
1. A solitary swelling.
2. Multiple lipomatosis : in which the limbs or the trunk are the seat of multiple lipomas **(differential diagnosis of multiple swellings).**
3. Diffuse lipomatous deposits : These can occur in certain areas, e.g. patients with myxedema have supraclavicular fatty deposits, elderly persons may develop lipomatous deposits below the chin and sometimes females may develop painful fatty deposits in the thigh **(Dercum's disease).**

[C] According to the site of origin : Solitary lipomas are classified into :
1. Subcutaneous lipomata : These are the commonest and they have the following characters :
(a) Slowly growing tumor in the subcutaneous tissue.
(b) The tumor is painless and is not tender.
(c) **Lobulated surface**, which may be attached to the skin at multiple points.
(d) The consistency is **soft**, although some lipomata may give **pseudofluctuation**; this is due to mobility of the tumor in its bed and because fat at warm temperature may undergo liquefication.
(e) It has a well-defined **slippery edge** due to movement of the tumor inside its capsule.
(f) The swelling is mobile over deep structures.

2. Subfascial lipomata : Occur under the deep fascia.
3. Intermuscular lipomata : Difficult to diagnose and has to be differentiated from a fibrosarcoma. The latter is hard in consistency and grows rapidly.

4. Submucous lipomata : A submucous lipoma in the larynx may cause respiratory obstruction. A submucous lipoma in the intestine may initiate intussusception causing intestinal obstruction.

5. Parosteal lipomata : Arise in relation to cranial bones and cause erosion of the underlying bones.
6. Extradural lipomata : Are sometimes found within the spinal canal and may cause paraplegia (never in the skull, no fat).

7. Intra-articular lipoma.
8. Retroperitoneal lipoma.

Complications : Are rare and include :
1. Degenerative changes leading to liquefication and calcification.
2. Malignant transformation is very rare, but it can occur only in a retroperitoneal lipoma.
3. Submucous lipoma and extradural lipoma (see before).

Treatment : Surgical excision.

Callosity

Area of superficial skin thickening due to continuous friction and pressure, usually not painful.
Treatment : should avoid the predisposing factor and it should be shaved with a razor blade and repeatedly painted with salicylic acid.

Corn

Similar to callosity but caused by pressure as well, compressing the sensory nerve endings causing **pain** with similar treatment.

Wart

Due to viral infection in an abrasion causing localized overgrowth of the epidermis and papillae of the skin. It is small horny projection and may be multiple. Its treatment includes :

1. Curettage & diathermy.
2. Repeated application of glacial acetic acid.
3. Cryosurgery.

Papilloma

Benign tumor, usually pigmented commonly develops in large numbers in the face, arms and upper trunk. Treatment is excision, cryosurgery or cautary.

Hypertrophic scar

- It is an increase of both intensity and duration of the active phase of scar formation.
- It is common in young ages after burns.
- The scar is very thick, red, itchy for 3 to 6 months then the condition regresses gradually
- **Treatment :** Excision & Plastic repair.

Keloid

- A rare condition, commonly over the sternum.
- There is inhibition in maturation and stabilization of collagen fibers.
- The same picture as hypertrophic scar but keloid may spread among nearby healthy tissues and continues to get worsen even after years.
- High rate of recurrence after excision or X-ray therapy.
- **Management :**
  1. Shaving of excess tissue then resurfacing by thin skin graft.
  2. Steroid injection.

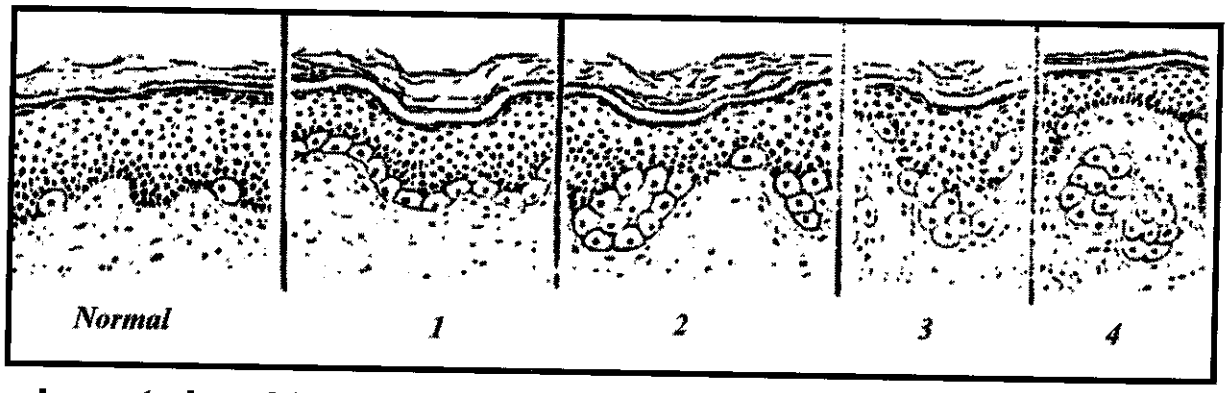
Items	Hypertrophic scar	Keloid
Definition	Delayed maturation of collagen fibers in a wound.	Failure of maturation of collagen fibers in a wound.
Induration	Limited to the scar.	Extends beyond the scar.
Course	Stationary after 6 months.	Progressive even after 6 months.

Melanocytic Tumors

• Melanocytes lodge in between the basal cells of the epidermis. Various hamaratomas or true tumors can arise from the melanocytes.

Benign lesions of melanocytes :

1. Lentigo : Melanocytes replace the basal layer of the epidermis over a certain area.
Clinically : It appears as a spot varying in color from pale brown to black.
2. Junctional-pigmented nevus : More proliferation of melanocytes at the junction of epidermis and dermis.
Clinically : Looks like a lentigo.
3. Compound pigmented nevus : Some melanocytes pass to the dermis. These melanocytes lose their capacity to form melanin and they are called nevus cells.
Clinically :
• Lesions are raised above the surface and vary in color from pale brown to black.
• Sometimes dark hairs are seen growing from the surface of the lesion. Rarely, an extensive area of the skin is replaced by such hairy-pigmented nevus **(Giant hair nevus).**
4. Intradermal pigmented nevus : The commonest.
Clinically : They look like compound pigmented nevus.



Can pigmented nevi turn malignant ?

1. The giant hairy-pigmented nevi can give rise to metastases.
2. After puberty any pigmented nevus with junctional activity has the potential to undergo malignant changes, but this is rare.

Indications for surgical excision :

1. For cosmetic reasons.
2. If they are subjected to repeated trauma e.g. during shaving.
3. If there is suspicion of malignant transformation.

Adequate surgical excision should be performed and the specimen should be examined histologically.

Precancerous skin lesions

Squamous keratosis (actinic or senile) :

Present by dry, rough, inelastic and irregular pigmented areas of skin with hyperkeratosis. It is the most important precursor of squamous cell carcinoma.

Bowen's disease :

Arises in non exposed skin with sharply demarcated, rounded reddish patches which enlarge slowly with marked cellular hyperplasia.

Melanoma

Incidence :

• In Western countries the incidence of malignant melanoma is increasing, which may be due to defective ozone layer. Malignant melanoma is almost unknown before puberty.

Etiology :

1. Prolonged exposure to ultraviolet rays of the sun.
2. Albinism and xerodermia pigmentosa.
3. On top of a benign nevus (discuss).

Criteria of malignant transformation of a benign nevus :

1. Increase size or volume of the tumor.
2. Increased pigmentation or depigmentation.
3. Ulceration and bleeding.
4. Pain.
5. Induration.
6. Surrounded by halo of pigmentation or multiple satellites.

Pathology and clinical types :

Items	Superficial spreading melanoma	Nodular melanoma	Lentigo melanoma	Acral lentiginous melanoma	Amelanotic melanoma
Incidence	64 %	12-25 %	1-15 %	Rare	Rare
Site	In any part of the body.	In any part of the body.	Usually in the face.	In the palms and soles. It may occur beneath the nail.	
Age	Usually in middle age.	In younger age groups.	Elderly persons.		
Clinically	1. Raised above the surface. 2. Edge : Irregular.	1. Raised above the surface. 2. Surface : Smooth. 3. Color : Gray or Black. 4. Liable to ulceration.	Some areas may regress.		It is not pigmented.
Prognosis		Bad.	The least malignant type.	Poor.	Very poor.

• Malignant melanoma may rarely arise in the eye, in the meninges, or at the mucocutaneous junction as the anal canal.

Factors, which have a major influence on the prognosis, are :

[A] The thickness of the tumor **(Breslow classification).**

[B] The depth of invasion of the skin **(Clark's level of invasion)** as follows :

1. Epidermis.
2. Dermo-epidermal junction.
3. Superficial papillary dermis.
4. Deep papillary dermis.
5. Subcutaneous tissue.

Spread :

1. Direct : To subcutaneous tissues and the deep fascia.

2. Lymphatic : By permeation or embolism. Lymphatic permeation leads to satellite nodules around the tumor or between the tumor and the regional lymph nodes.

3. Blood : Malignant melanoma can spread to any organ. Secondary deposits are usually black.

Differential diagnosis:

1. Granuloma.
2. Pigmented basal cell carcinoma.
3. Hemangioma.
4. Compound or junctional nevus.

Investigations :

- The only sure method of diagnosis of malignant melanoma is **Biopsy & Histological Examination.**
- The biopsy should include the whole skin & S.C. tissue with safety margin 3mm at least.

Treatment :

1. The primary lesion : Should be excised with a safety margin 2-3 cm together with the subcutaneous fat but not including the deep fascia.

- If the tumor thickness < 1mm → safety margin is 1 cm.
- If the tumor thickness 1-4mm → safety margin is 2 cm.
- If the tumor thickness > 4mm → safety margin is 3 cm.

2. The lymph nodes :

- If the regional lymph nodes are enlarged and firm, a radical block dissection is performed.
- If the lymph nodes are not frankly malignant on clinical examination, FNAC is performed.
- Prophylactic block dissection is no longer performed.

3. Metastases : Are treated by chemotherapy, interferon and by interleukin-2.

Items	Basal cell carcinoma (Rodent ulcer)	Squamous cell carcinoma (Epithelioma)
Definition	• This is a locally malignant lesion, which arises from the basal cells of the epidermis or from the equivalent cells of hair follicles and sweat or sebaceous glands.	• Malignant tumor of stratified squamous epithelium.
Incidence	• The disease is more common in males above the age of 40 years. It is the commonest malignant lesion of the skin.	• The tumor is more common in males especially elderly persons.
Etiology	1. Prolonged exposure to ultraviolet rays of sun (most important). 2. Persons with a light colored complexion are more liable (presence of melanin pigment protects against the effects of ultraviolet rays). 3. Albinism and xerodermia pigmentosa predispose to multiple basal cell carcinomas all over the body. 4.patient receiving immunosuppression	1. Prolonged exposure to ultraviolet rays of the sun. 2. Previous irradiation. 3. Albinism and xerodermia pigmentosa. 4. Long standing irritation of the skin as : Chronic ulcers, sinuses, and old burn scars. A carcinoma arising on top of a scar is called Marjolin's ulcer. 5. Carcinogenic agents as : polycyclic hydrocarbons, coal tar derivatives, or mineral oils.
Pathology : **1. Site**	• 90 % of the lesions occur in the face especially above a line from the lobule of the ear to the angle of the mouth. • The commonest sites are the outer or inner canthus of the eye and the nasolabial fold.	• The tumor occurs more in the upper part of the face, the lower lip, and the dorsum of the hands. • Sometimes, the tumor arises at the mucocutaneous junctions.
2. Macroscopic picture	**1. The lesion usually starts as a small nodule** covered by thin epidermis. The nodule ulcerates after sometimes with serous discharge and bleeding. Healing sometimes occur in one area of the lesion while further ulceration occurs. **2. Later the patient presents by an ulcerated mass :** • **The edge :** rolled-in and beaded. • **Size :** Small. • **The floor :** red & granular, and often covered with a dry crust or scab. • **The base :** Soft with late fixation to underlying structures. **3. Excavating type :** Where the ulcer erodes deeply into the underlying structures leading to destruction of the nose and infiltration of the nasal sinuses. **4. Other less common types include :** the cystic, the pigmented (containing melanocytes) or the flat superficial spreading type resembles psoriasis or eczema and presents as a red scaly patch.	• **An ulcerated mass (malignant ulcer) :** **1. Size :** Grows rapidly, may reach large size. **2. The edges :** Are raised and everted. **3. The floor :** Malignant fungating tissue. **4. The base :** Is indurated and it becomes rapidly fixed to the underlying tissues. **5. Secondary infection :** May occur and there may be blood stained discharge.

3. Microscopic picture	• The tumor cells are arranged so as the outer layer of these groups of cells is composed of low columnar cells arranged side by side like **a palisade.** • Inside this layer the cells are polyhedral with large basophilic nuclei. • There is no tendency to keratinization. • The stroma shows infiltration by lymphocytes.	• In well-differentiated tumors each mass shows the same layers as the normal stratified squamous epithelium with keratin inside and basal cells outside. • These cell masses with central keratin are called cell nests (epithelial pearls). *Presence of keratin & prickle cells indicates the differentiated nature of the tumour. Undifferentiated tumors, cells are anaplastic with no cytological evidence of their origin* • Broder's classification is used in squamous cell carcinoma and depends upon the number of cell groups with cell nest appearance : **Grade I :** 75-100 % Keratinized groups **Grade II :** 50-75 % Keratinized groups **Grade III :** 25-50 % Keratinized groups **Grade IV :** 0-25 % Keratinized groups
4. Spread	**1. Direct spread :** To the surrounding and underlying structures. **2. Lymphatic spread :** Does not occur. If the draining lymph nodes are enlarged, this is usually due to secondary infection or the presence of a focus of basosquamous carcinoma. **3. Blood stream spread :** Does not occur.	**1. Direct spread :** The tumor rapidly infiltrates the adjacent structures. **2. Lymphatic spread :** To the regional lymph nodes. Sometimes the nodes are enlarged as a result of secondary infection. In patients with Marjolin's ulcer, the fibrosis associated with the scarring makes lymphatic dissemination late. **3. Blood stream spread :** Is very uncommon.
Differential diagnosis	1. Basal cell carcinoma. 2. Squamous cell carcinoma. 3. Malignant melanoma. **4. Keratoacanthoma :** It commonly occurs due to sun exposure, in the face or hands of middle aged people as a firm rounded red papule, which may grow to 2 cm or more in a few weeks. The swelling often ulcerates & may undergo spontaneous healing in 6 monthes. • **Treatment :** Surgical excision is recommended to permit adequate histological examination.	
Investigations	• Diagnosis should be confirmed by a biopsy. • If the lesion is small, excision biopsy is performed; otherwise a small piece of the edge with an area of adjacent skin is excised.	

Treatment

[A] Radiotherapy :

- ✓ Basal cell carcinoma is very radiosensitive.
- ✓ The dose of radiotherapy is fractionated over several weeks to diminish the scaring and necrosis.

Contraindications :

1. If there is involvement of an underlying cartilage or bone
2. If the lesion is near the eyes

[B] Surgery Is indicated for :

1. **Small lesions.**
2. **Infiltration of underlying cartilage or bone :** Because :
   (a) The bone cells will protect the malignant cells from the effect of radiotherapy.
   (b) Radiation is likely to cause necrosis of involved bone & cartilage
3. **Radio resistant lesions.**
4. **Recurrences after previous radiotherapy.**

- Surgery should include an **adequate safety margin** ½ cm at least regarding the surface and the depth.
- The pathologist should check that an adequate safety free margin has been excised, and if not, **revisional surgery** or **radiotherapy** should be applied.
- **The defect created after excision** can be closed by primary suture or by various plastic rotational flaps.

[C] Other possible lines for treatment include :

1. **Cryosurgery.**
2. **Local application of cytotoxic drugs :** as 5-flurouracil ointment.

- **Prognosis :** Excellent and provided that the lesion has been excised completely, the cure rate is 100 %.
- **Recurrence** of the lesion is due to leaving behind foci of malignant tissue and this is more liable to occur if there is infiltration of bone or cartilage.

Two modalities of treatment are available :

[A] Surgery Is indicated for :

1. **Small lesions :** Treated by excision biopsy with an adequate safety margin.
2. **Infiltration of underlying cartilage or bone.**
3. **Radioresistant lesions.**
4. **Recurrences after previous radiotherapy.**
5. **Marjolin's ulcer.**
6. **Block dissection of metastatic lymph nodes.**

- The safety margin for epithelioma is at least 2 cm except in the face where it is 0.5 inch.

[B] Radiotherapy :

The main indication of radiotherapy is for tumors of the head and neck particularly for poorly differentiated lesions.

- **Prognosis :** 90 % five years cure rate.

Hand Infections

Hand infections

Due to the use of the hand and its exposure to contamination, the frequency of hand infection is high prevalent among manual workers and house wives.
Proper treatment and early interference is needed to preserve the function of the hand.

Classification :

[1] Cutaneous and subcutaneous infections :

1. Pulp space infection. 2. Subcuticular and subcutaneous whitlow.
3. Web space infection. 4. Paronychia.

[2] Fascial spaces infection :

1. Midpalmar space infection. 2. Thenar space infection.
3. Space of Parona infection. 4. Hypothenar space infection.

[3] Synovial sheaths infection :

1. Acute digital tenosynovitis. 2. Ulnar bursitis. 3. Radial bursitis.

[4] Bone and joint infections.

In the management of hand infection take care of the following points :

1. General and local rest to the hand in the elevated position.
2. Early administration of antibiotics.
3. Early drainage of infection i.e. do not wait for fluctuation.
4. Incision must be planned and under general anesthesia.
5. Incision is done under tourniquet to have the field dry to explore the abscess.
6. Substance of the drain must be soft e.g. piece of rubber glove. (The drain must be outside tendon sheath).
7. The hand must be maintained in the elevated position to prevent edema and congestion.
8. Early restoration of function by movement.
9. Position of immobilization is that of function of the hand.

Subcuticular and subcutaneous whitlow

- It is subepithelial collection of pus.
- Drainage is done by excision of the insensitive roof without anesthesia.
- It may be part of collar-stud abscess, with SC component, which requires anesthesia for its evacuation.
- A subcutaneous abscess may also form without a subcuticular component.

Infections related to the nail

Peri-ungual infection = Paronychia

I- Acute paronychia (subcuticular and subcutaneous **whitlow**):

- Infection occurs in the nail fold and may extend to the nail bed.
- The commonest of hand infection.

Complications :

1. Extension subungual, i.e. below the nail.
2. Extension to pulp space.
3. Lymphangitis and lymphadenitis.

Clinical picture : Localized pain and tenderness to one side of the nail fold accompanied with small swelling, which is red in color.

Treatment :

1. Infection localized to one side of the nail fold : An oblique incision over the tenderest point is done. This incision must extend up to the edge of the nail fold.

2. If infection extends all round the nail fold : 2 Incisions are done at the angles of the nail fold and the proximal wedge is reflected.

3. If infection has extended to the subungual space : Two incisions are done at the angles of the nail fold and the proximal wedge is reflected and the proximal ½ of the nail is removed.

3. If infection extends below the nail and pus is seen below it (floating nail) : The nail becomes a dead nail and it must be removed to drain infection.

II- Chronic Paronychia :

- Is common in washerwomen and may be non-specific chronic paronychia or due to fungus infection.
- It usually affects more than one finger.

Treatment :

- Antifungal treatment by local dyes and, systemically by gresofulvin.
- Removal of the affected nail.

Pulp space infection

Anatomy of the pulp :

- The pulp is the distal subcutaneous space infront of the bone of the phalynx.
- Proximally this space is shut from the middle pulp space by a transverse septum attached to the bone.
- The space is divided into multiple loculi by antro-posterior septa attached from the skin to the phalynx. These spaces contain some fat.
- The digital artery gives a branch to the proximal ½ of the bone before it enters the space.

Pathology of pulp space infection :

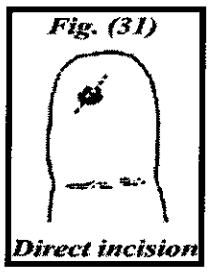
- **Causative organism** is mainly Staph and to a less extent Strept.
- **Route of infection :** direct inoculation by a pinprick, extension from paronychia, extension from middle pulp space, extension from cuticular, or subcuticular infection.
- Infection occurs on the pulp and as the space is limited the tension rises rapidly and infection proceeds quickly.
- Obliteration of the blood supply occurs. Sequestration of the distal ½ of the bone may occur. The proximal part of the bone is protected as its blood supply comes from the digital artery before it enters the space.

Clinical picture :

1. **Localized pain and tenderness** over the distal pulp space.
2. **Swelling** which is indurated and red.
3. **General manifestations of infection.**
4. **No tenderness over the finger proximally or in the palm** (D.D. of tenosynovitis).
5. **Range of painless movement in all the joints** (D.D. of arthritis).

Complications :

1. Osteomyelitis of the distal phalanx.
2. Extension to the middle pulp space.
3. Teno-synovitis. 4. Arthritis.
5. Blood stream and lymphatic spread.



Treatment :

1. Do not wait for fluctuation.

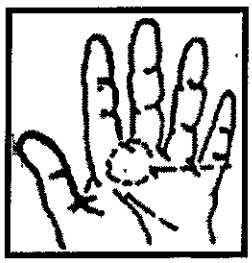
2. The incision is sited directly over the most tender point and in direction of Langer's lines.

Web space infection

Anatomy of the web :

• The heads of the metacarpal bones are attached together by the transverse palmar ligaments. The web space lies infront of these ligament and deep to the skin. The space communicates proximally with mid palmar space. Distally it communicates with proximal pulp space of the adjacent fingers. It is filled with fat and is crossed by the lumbrical muscles.

Clinical Picture :



1. The patient complains of **hot, red, tender swelling** at the site of a web.
2. The fingers cannot be approximated because adduction increases pain.
3. The movement of all joints of the fingers is free (DD septic arthritis).
4. No swelling in fingers (DD Teno-synovitis).

Complications : Extend to :
1. Mid palmar space. 2. Proximal pulp Space. 3. Tendon sheath.

Management :

1. A transverse skin incision is made over the point of maximal tenderness or fluctuation, and then an artery or a sinus forceps are opened in a longitudinal direction (to avoid damage to the digital nerves and vessels), through the subcutaneous tissue to enter the abscess cavity.
2. Vertical dorsal incision.

Fascial spaces

Anatomy of Fascial Spaces of the Hand :

The palmar aponeurosis is triangular in shape. Its apex is proximal and into it is inserted palmaris longus. Distally it gives slips to be attached to the transverse palmar ligaments between the medial 4 fingers.

• The medial edge of palmar aponeurosis gives a septum, which dips backwards and is attached to 5th metacarpal bone. Medial to this septum is the hypothenar space containing short muscles of little finger.

• Another septum extends from the lateral edge to the anterior aspect of the 3rd metacarpal bone. This septum separates laterally the thenar space from the mid palmar space. This septum also covers the adductor pollicis muscle.

• Mid palmar space is further divided into a superficial compartment superficial to the tendons and deep to the palmar aponeurosis called subaponeurotic space. The space behind the flexor tendons is called retro tendenious space.

Hypothenar space infection

Source of infection :

1. Direct inoculation. 2. Complication of teno-synovitis of the flexor tendon of the little finger.
3. Extension from the web between little and ring fingers. 4. Extension from proximal pulp space.

Causative organism : Staph., Strept. ...etc.

Clinical picture :

1. Localized pain and tenderness with hotness and redness in the hypothenar region.
2. Swelling on the medial aspect with accentuation of the concavity of the hand.
3. Free range of painless movements in the inter-phalyngeal and metacarpophalygeal joints of little finger (D.D. septic arthritis, teno-synovitis, ulnar bursitis).

Complications :

• Extension to web space, proximal pulp space, mid palmar space or tendon sheath.
• Lymphatic and blood stream infection.

Management :

• A Vertical incision at the site of maximal tenderness or along the medial border of the fifth metacarpal bone is done. The space is entered by a sinus forceps i.e. Hilton's method.

Thenar space infection

Source of infection :

1. Direct inoculation.
2. Extension from web space.
3. Extension from proximal pulp space.
4. Complication of tenosynovitis.

Clinical Picture :

- **Swelling in the thenar eminence** which is hot, red, and severely tender.
- **Edema on the dorsum of the hand.**

Management :

- General principles are followed.

Incision :

1. Transverse incision at the web then the space is opened by a sinus forceps (Hilton's method).
2. Vertical incision on the lateral aspect of the back of the 2nd metacarpal bone, this is the incision commonly used.
3. An incision that is done along the medial side of the thenar eminence should stop 2cm distal to the distal wrist crease to avoid injury of motor branch of the median nerve that supplies the thenar muscles.

Mid palmar space infection

- The space is bounded anteriorly by palmar aponeurosis, medially by medial septum separating it from the hypothenar space, laterally by the lateral septum separating it from the thenar space.
- Mid palmar space is further divided into subaponeurotic and retro-tendenous spaces by flexor tendons.

Source of Infection :

1. Web space infection.
2. Tenosynovitis.
3. Thenar and hypothenar infection.
4. Direct inoculation.

Clinical Picture :

1. Marked swelling which obliterates the palmar concavity (Frog's hand).
2. The middle of the palm is hot, red and tender.
3. Dorsal swelling is much more apparent than in any other hand infection.
4. Pain on moving the fingers but there is a free range of movement of these fingers.
5. Sometimes a subaponeurotic infection forms a subcutaneous loculus by forming a hole in the palmar aponeurosis **i.e. Collar-Stud Abscess** is formed. It is important to exclude deep sub-aponeourotic infection when one is draining a subcutaneous abscess in the palm.

Complications :

1. Infection of tendon sheaths.
2. Spread to the web.
3. General spread.

Treatment : Incision when pus is formed : either :

- If there is a collar-stud abscess, incise along one of the hand creases, evacuating the subcutaneous abscess. The hole in the aponeurosis is explored and is enlarged to drain sub-aponeourotic collection.
- A transverse incision is done like that of a web space and by Hilton's method a sinus forceps is introduced into the retrotendenous space to drain pus.

Infection of the space of Parona

This space lies in the distal part of the forearm between the pronator quadratus and the flexor muscles. Drainage should be along the ulnar side of the forearm.

Teno synovitis

Surgical anatomy of the tendon sheathes :

1. The middle 3 fingers are surrounded by three tendon sheathes which extend from the distal phalanx to the level of the head of the corresponding metacarpal bone.
2. Tendon sheath of the thumb extends in the palm and lower part of forearm forming the radial bursa.
3. Tendon sheath of the little finger communicates with the sheath surrounding the flexor tendons in the palm. The point of meeting between the sheath of the little finger and the common flexor sheath is called ***(Kanavel point).*** The common flexor sheath extends up to the lower part of the forearm.

Source of infection :

1. Direct inoculation by pricks and wounds.

2. Extension from a nearby infection :

- Pulp space infection.
- Web space infection.
- Mid palmar space infection.
- Arthritis and osteomyelitis.

3. Blood stream infection.

Pathology :

1. Synovitis with pouring of excessive synovial fluid, which is clear at the start.
2. The fluid later becomes turbid due to the formation of pus.
3. The sheath is distended with the fluid.
4. The nutrition of the tendon is impaired.

Complications :

1. Sloughing of the tendon.
2. Adhesions between the visceral and parietal layers of the sheath.
3. Stiffness in the fingers and loss of movements.
4. Spread to the fascial spaces in the palm and forearm.
5. Blood stream spread, Bacteremia, septicemia, toxemia.

Clinical picture :

1. History of the causative agent.

2. General manifestations of inflammation : high temperature, tachycardia, malaise, headache, rigors, constipation, white-coated tongue.

3. Swelling :

- There is symmetrical enlargement of the palmar aspect of the finger extending from distal phalanx to the head of the metacarpal bones. The finger will be in the semi-flexed position.
- The swelling in case of little finger extends in the middle of the palm and lower part of forearm.
- Swelling of the thumb extends in the thenar eminence and lower part of forearm.

4. Hotness and redness over the swelling.

5. Tenderness over the tendon sheath but maximal at the cul-de-sac i.e. at the distal phalanx and at the head of the metacarpal bone.

6. In the little finger the tenderest point is the Kanavel point. This point is between the 2 transverse palmar creases opposite the head of the 5th metacarpal bone.

7. Pain is increased on moving the finger, and is exaggerated by passive extension of the finger.

Management :

1. Early proper diagnosis.
2. Full antibiotic therapy.
3. If the case is diagnosed early, aspirate the synovial fluid at the proximal culde-sac and inject crystalline penicillin.
4. The affected sheath is exposed and drained at the site of the causative injury and also at its proximal and distal extremities. The sheath is exposed by short transverse incisions. In the case of the thumb and little finger this may necessitate an incision in front of the wrist to expose and drain the radial and ulnar bursae. After drainage the sheath is washed thorough with a solution of penicillin introduced by means of a whistle-tipped ureteric catheter and the wounds are left open.

Muscle & Tendon

مكتبة ادهم 01006068182

Diseases of Muscles, Tendons & Fascia

Carpal tunnel syndrome

- The carpal tunnel is formed by the flexor retinaculum anteriorly and by the distal row of the carpus posteriorly. The median nerve and flexor tendons pass through it.
- Swelling of synovial sheaths of flexor tendons increase the bulk of the contents of the tunnel compressing the median nerve.

Etiology :

- This disorder affects essentially the middle aged females.
- Precipitated by : Rheumatoid arthritis, myxedema, pregnancy or as stenosing teno-vaginitis.
- May be idiopathic.

Clinical picture :

- **Early manifestations**
  1. Pain in the thumb & lateral three fingers that gets worse by night.
  2. Pain is increased by fully flexing the wrist.
  3. The little finger is never affected.
  4. The palm is not affected as the palmar branch of median nerve escapes compression.
  5. Relief of pain is obtained by injecting the tunnel with lidocaine & corticosteroids and by splinting the wrist at night.
- **Late manifestations**
  1. Paraesthesia and altered sensibility of skin supplied by the median nerve.
  2. Weakness and atrophy of the thenar eminence muscle.

Treatment : Longitudinal incision of flexor retinaculum.

Dupuytren's contracture

- It is a localized thickening of the palmar fascia. It is known as palmar fasciitis.
- The fascia thickens to form nodules and contracts and withdraws the fingers into flexion. It starts at the base of little finger, and later it involves the ring finger, less often the middle and the index fingers (partial or less commonly complete claw hand).
- commonly confused with the hand pain produced by cervical spondylosis.
- It has a higher incidence in cirrhotics, alcoholics, epileptics under phenytoin treatment and diabetics

Treatment :

1. Early cases can be treated by night splintage and gentle stretching.
2. Surgical excision.

Simple ganglion

- It is a cystic swelling related to a joint or tendon sheath, occurring most often on the dorsum of the wrist and foot.
- The cyst contains clear gelatinous fluid and its wall consists of fibrous tissue.
- It is either due to herniation of synovial membrane through tendon sheath or mucoid degeneration predisposed to by trauma.

Clinical picture :

Rounded cystic swelling, which is painless and not attached to skin. It is translucent, movable when tendon is relaxed and becomes fixed on contraction.

Treatment :

1. Subcutaneous rupture by pressure.
2. Puncture followed by pressure.
3. Puncture followed by injection of hyaluronidase.
4. Excision through a transverse incision.

Compound palmar ganglion

• This is TB synovitis of the synovial sheath of the fingers under the flexor retinaculum. It contains TB granulation tissue, free fluid and melon seed bodies (fibrinous).

Clinical picture :

A fluctuating swelling in the lower part of forearm and palm with a band like constriction at the level of the flexor retinaculum.

Treatment :

1. Anti-TB treatment.
2. Immobilization in plaster of paris.
2. Surgical excision : Complete excision is not recommended and incision with evacuation of contents is preferred.

Volkmann's Ischemic Contracture

(Contracture = permanent shortening of the muscle due to its replacement by fibrous tissue)

Etiology :

• Injury to the arterial supply of the muscles of the limb leads to ischemia & gradual death of these muscles & its replacement by fibrous tissue. This is common around the elbow & the knee.

Clinical picture :

Early : Manifestations of acute ischemia in the form of **P**ain, **P**allor, **P**aralysis, **P**arasthesia, **P**ulselessness.

Later : The patient will present with the typical deformity of : flexion at the wrist joint, extension at the metacarpophalyngeal joins, flexion at all the inter-phalangeal joints. Extension of the wrist leads to more flexion at the interphalangeal joints. With flexion at the wrist, the surgeon can passively extend the finger.

Treatment of Volkmann's Contracture :

1. Prophylactic treatment : If the patient develops acute ischemia with the **5(Ps)** of **P**ain, **P**allor, **P**aralysis, **P**arasthesia, **P**ulselessness. The plaster is removed and the fracture is recorrected, and the patient is given vasodilators. If no immediate response is obtained, the vessel is exposed and dealt with according to the type of injury present.

2. In established case :

(a) Early : Do passive traction of the shortened muscles in the hyper extended position with splintage in this position.

(b) Late : Sliding the origin of the flexor muscles from the medial epicondyl downwards to the ulna, extra length is given to the shortened muscles correcting the shape but not the function of this hand.

Chronic tendonitis

1. Tennis elbow:

The patient complains of pain in the elbow at rest and in particular when he uses the hand (extensors). Pain develops at the site of attachment of the extensor muscles of forearm to the lateral epicondyle.

2. Golfer's elbow:

Similar to tennis elbow, but it affects attachment of flexor muscles of forearm to the medial epicondyle.

3. Supraspinatus tendinitis:

Pain is felt in the shoulder especially on abduction and rotation. There is localized tenderness over the insertion of the supraspinatus tendon.

4. Plantar fasciitis:

Pain and localized tenderness occur under the heel at site of attachment of plantar fascia to the os calcis.

- **Treatment :**

(a) Ask the patient to avoid powerful contraction of the involved muscles.
(b) Local injection of hydrocortisone and local anesthetics.
(c) When supraspinatus tendon is calcified surgical release or decompression of tendon may be needed.

5. Stenosing teno-vaginitis :

Spontaneous thickening of tendon sheath, which gradually entrap the tendon.

- **Dequervain's disease :** The common sheath of abductor pollicis longus and extensor pollicis brevis tendon at wrist is affected by stenosing thickening. Patient develops pain with movements of the thumb.
- **Trigger finger :** Stenosing thickening affects the retinacula of the flexor tendons in the palm. The affected finger can be flexed, but on extension the patient feels difficulty at a certain angle after which the finger snaps extended like a trigger.
- **Treatment :**

(a) Local injection of hydrocortisone.
(b) Stenosing retinaculum can be divided surgically.

Chronic bursitis

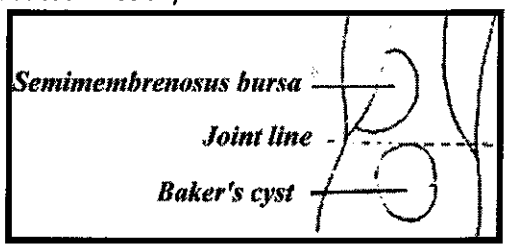
Bursae are fluid filled cavities.

- **Anatomical bursae :**

Are normally found to allow easy movement between tendons, bones, and skin.
Chronic inflammation of such bursae usually results from repeated friction, and presents with mildly painful cystic swellings.

Common sites :

1. Prepatellar bursitis (House-maid's knee).
2. Infrapatellar bursitis (Clergyman's knee).
3. Olecranon bursitis (student's elbow).



- **Semimembranous bursitis :**

The bursa lies in the popliteal fossa between the medial head of gastrocnemius and semimembranosus tendon and frequently communicates with the cavity of knee. The bursa is rounded fluctuating swelling, becomes tense on extension and flaccid on flexion of the joint.
Treatment : Careful dissection and excision.

- **Adventitious bursae :**

> Develop in any site of friction between two layers of tissues. A known example is the **"bunion"** which develops between the skin and the head of the first metatarsal, particularly with the hallux valgus deformity.

> If friction can be abolished, bursitis may regress, otherwise the treatment is excision and correction of any underlying deformity.

Swellings of Popliteal Fossa

[A] Cystic swellings :

1. Skin and S.C. tissue : Abscess, hemangioma, lymphangioma.
2. Vein : Saccular varicosity of upper end of short Saphenous vein, V.V.
3. Artery : Popliteal aneurysm, arterio - venous fistula.
4. Semimembranosus bursitis.

[B] Solid swellings :

1. Skin and S.C. tissue : Lipoma, neurofibroma.
2. Muscles : Fibrosarcoma.
3. Nerves : Neurofibroma.
4. Periosteum : Fibrosarcoma.
5. Bones : Acute and chronic osteomyelitis, osteoclastoma, osteosarcoma, secondaries of lower end of femur or upper end of tibia.

Ingrowing toenail

Incidence & Definition :

- A common problem in which a sharp edge of the big toe nail ingrows in the adjacent skin fold.
- Young males predominate.

Predisposing factors :

1. Faulty nail trimming oblique.
2. Tight shoes. Trimming of the nail sides may leave behind a sharp spike that starts the condition.

Treatment :

[A] Conservative treatment indicated in early cases :

- The aim is to lift off the nail spike : A piece of gauze soaked in an antiseptic is inserted beneath the ingrowing part of the nail.
- The piece of gauze is changed daily.
- The patient is instructed to avoid tight shoes, and is shown the proper technique of square nail trimming.

[B] Surgery :

- Indicated for failure of conservative treatment, and for suppurative cases.

Wedge resection :
Position: On the back.
Tourniquet : On toe near its base.
Anesthesia : General or local (without adrenaline).

- A longitudinal incision is made through the affected side of the nail down to the bone and extending proximally to the nail root.
- Another incision is made through the skin by the side of the lesion down to the phalanx
- The wedge of tissue between the two incisions is excised, leaving bare bone without any remnant of the germinal matrix.
- The gap may be left open to granulate or may be closed with 2 or 3 interrupted sutures

Soft tissue sarcomas

Definition :
Malignant tumors arising in the extra-skeletal connective tissue.

Etiology : Not exactly known :
1. Irradiation.
2. Lymphangiosarcoma may develop in the chronic post mastectomy arm edema **(Stewart-Treves disease).**
3. Neurofibrosarcoma may develop in patients with **Von Recklinghausen's disease.**

Pathology : Types :
1. Malignant fibrous histocytoma.
2. Liposarcoma.
3. Rhabdomyosarcoma.
4. Synovial sarcoma.
5. Malignant nerve sheath tumor.
6. Leiomyosarcoma.
7. Fibrosarcoma.
8. Angiosarcoma.

Gross appearance :
There is a well-defined pseudocapsule. Enucleation of the tumor from within the pseudocapsule is inadequate. The cut section is fleshy & shows areas of necrosis & hemorrhage.

Spread : direct, blood & lymphatic spread.

Clinical picture :
1. Swelling.
2. Pain & disability are often absent.
3. Spread.

Investigations :
1. Open biopsy.
2. CT & MRI.
3. Investigations to detect spread.

Treatment :
1. Operable cases :